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## Table of Contents





### SOME NOTES ON NEURO-SURGERY.[1]

By LEONARD C. E. LINDON, M.S. (Adelaide), F.R.C.S. (England),
*Honorary Assistant Surgeon, Adelaide Hospital, Adelaide.*

THIS paper is composed from notes made while visiting some of the neuro-surgical clinics of England and North America; the methods to be discussed are in no way new, but I had seen little or nothing of them until this year, and perhaps they may be of as much interest to you as they were to me. We labour under a distinct handicap in Australia so far as the surgery of special regions is concerned, owing to the small and scattered population. In no branch of surgery is this handicap greater than in neuro-surgery; there is not sufficient concentration of material—at least as regards South Australia—to make it possible to organize the team work which is so noticeable a feature of the clinics abroad and which has contributed so largely to the excellence of the work done in them. A tremendous amount of time must be spent over each case and the work requires the services of a large staff of specially trained clinical and pathological assistants. Until such a clinic can be established in Australia we shall not keep abreast of the advances being made in neuro-surgery in North America, England and the Continent. There is room for one such clinic in Australia, but I doubt if the numbers will ever justify the establishment of a neuro-surgical clinic in each capital. It will be of interest to many of you to know that Hugh Cairns has organized a proper team at the London Hospital and has certainly impressed upon his colleagues that vastly improved results may be obtained by strict attention to the methods of

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 27, 1930.

Professor Harvey Cushing, the undoubted leader in modern neuro-surgery.

I propose to discuss some of the more recently standardized adjuncts to clinical diagnosis, such as the injection of air for the purpose of the diagnosis of intracranial lesions and the use of manometry in lesions of the spinal cord. I wish to emphasize the point that nothing can replace thorough and frequently repeated clinical examinations as the basis of neurological diagnosis. I was very much impressed by the fact that the neuro-surgeons are all, first and foremost, diagnosticians and have trained themselves to be capable of complete examination of the organs of special sense, the lesions of which play such an important part in the diagnosis of intracranial disease. It is to be understood that the methods to be described should never precede a complete clinical examination, for the one reason that these methods are not entirely free from risk; a routine examination will often disclose very definite contraindications to the injection of air.

### The Use of Air in Diagnosis and Treatment.

Various radio-opaque fluids have been used for the purpose of outlining the surface and cavities of the brain by subarachnoid, ventricular and arterial injection. At the best of times the interpretation of the radiograph is difficult and open to many errors. I should think that the injection of the cerebral arterial tree, *viâ* the internal carotid, must be the least satisfactory, and the most danegrous, method, but I must admit that I have no experience of it.

Of the methods of air injection the subarachnoid injection by the lumbar route is the least dangerous and has the value of revealing both the ventricular and the cortical outlines. While it is the safest method, it certainly appears to be far more distressing at the time to the patient than is ventricular puncture, though recovery takes place in two or three hours and the mortality rate over a very large series is less than one *per centum*.

### *The Technique of Air Injection.*

The procedures about to be described should be performed in the operating theatre; they are major operations, carrying with them great risks if there is carelessness or if asepsis is not complete. Any procedure dealing with the subarachnoid space is deserving of the utmost precaution.

In doing an encephalogram lumbar puncture is first performed, the patient lying horizontal. The puncture should be made with the finest needle; even a very fine needle will leave a small hole in the dura and arachnoid membrane, through which cerebro-spinal fluid will leak for some hours, giving rise to headache and the possibility of infection. The cerebro-spinal fluid pressure is taken with a manometer; it is generally accepted that the normal lies between 150 and 200 millimetres of water. The cardiac and respiratory pulsations are observed and the response to compression of the jugular vein in order to exclude the presence of any "spinal block." The patient is then raised to a sitting position across the table; this position is maintained until the radiographs have been taken.

There are two methods used in the replacement of fluid by air: (i) The pressure for pressure method, controlling the replacement by the manometer, to insure that the final pressure does not exceed the initial pressure; (ii) the volume for volume method, which is simpler, and just as safe, provided one allows for the fact that the air is injected at room temperature, about 21·2° C. (70° F.), and will take up the brain temperature of about 37·8° (100° F.) and in so doing will expand by about ten *per centum*. Satisfactory radiographs should result if 120 cubic centimetres of fluid are replaced by 105 to 110 cubic centimetres of air; this is done by allowing ten cubic centimetres of fluid to escape and replacing it by slightly lesser amount sof sterile air and repeating the procedure until the requisite volumes have been obtained. The air is sterilized by drawing it into the syringe through sterile gauze or absorbent cotton. During the process the patient's head is frequently moved in all directions to insure a symmetrical distribution of the air. There is generally considerable reaction during the replacement. The air rises to the *cisterna magna*, whence it passes first to the various subarachnoid cisterns and intergyral spaces; the patient then complains of intense headache, chiefly frontal. After about thirty cubic centimetres of air have been injected air enters the fourth ventricle from the *cisterna magna* and passes through the third to the lateral ventricles. It is now definitely known that a "head station" for the sympathetic system is situated in the walls and floor of the third ventricle; the entrance of air into this ventricle is followed by intense sympathetic stimulation, with resulting tachycardia, dilatation of the pupils, profuse cold sweating, nausea, vomiting and collapse; a terrifying picture indeed if unexpected. Fortunately, this state does not last long and by the time the patient has returned to bed the only complaint is one of headache, which may last several days. I believe the patient should be kept in bed until the headache has passed off, though many medical practitioners perform encephalography in their consulting rooms and allow the patients to go home and take care of themselves—altogether too risky a proceeding. When there are post-traumatic arachnoid adhesions causing headache, the air replacement may break down these adhesions and give rise to signs of cortical irritation and œdema; a patient thus affected should obviously be in hospital under close supervision till the passage of time renders such complications unlikely. The technique of the subsequent radiography is not for me to describe, but I wish to say that the patient should be kept upright till the photographs have been taken.

Encephalography has been used mainly in the investigation of epilepsy and post-traumatic intracranial lesions, headaches, mental degeneration *et*

*cetera*. It is also of use in localizing cerebral neoplasms in the silent areas. A good encephalogram will give information concerning the patency of the normal subarachnoid channels, subarachnoid cysts or block, the deformity of the ventricles or hemispheres. Penfield, of Montreal, and Foerster, of Breslau, are jointly publishing a statement of the work of themselves and others on the subject of brain cicatrix and of the investigation and treatment of post-traumatic epilepsy, in which encephalography has proved so valuable.

### *Contraindications to Encephalography.*

There are three very definite contraindications to encephalography, as follows:

1. When there is evidence of an inflammatory intracranial lesions.

2. When there is any evidence of increased intracranial tension, in which the risk of causing a medullary pressure cone is obviously an absolute contraindication. It is a safe rule that lumbar puncture should never be performed until the optic discs have been examined and the presence of papillœdema has been excluded.

3. When the patient's general condition is such that the collapse associated with air replacement would not be tolerated or when the patient is so fat that he is unable to maintain the upright position.

If these precautions are observed encephalography is a relatively safe procedure which will frequently determine both diagnosis and localization when the ordinary routine methods have failed.

### Ventriculography.

Ventricular puncture can be done entirely under local anæsthesia, even in children. The trephine hole is made at a point six centimetres above the external occipital protuberance and three centimetres to one or other side. The best radiographs are obtained after both ventricles have been tapped; if only one ventricle be tapped it is very easy to fail to empty both ventricles; thus the subsequent replacement with air may be asymmetrical and a false apearance of asymmetry of the ventricular outlines results. The ventricle is tapped at the junction of the posterior and descending horns by passing the brain needle forwards and slightly downwards and outwards for a depth of four to six centimetres. I have been using Hugh Cairns's modification of Cushing's needle, a hollow, blunt-nosed, graduated needle, which is less likely to cause hæmorrhage or to damage the brain. The capacity of the normal ventricle varies within wide limits, but any ventricle which holds more than thirty cubic centimetres is definitely enlarged. The fluid removed is replaced by sterile air, but it is here even more imperative that the expansion of air be allowed for; if the ventricles be over-distended with air, the subsequent expansion of the air will tend to drive the mid-brain down into the hiatus of the tentorium, with tragic results unless the ventricles be immediately tapped and the pressure relieved. Air is absorbed rapidly from the subarachnoid space, but slowly from within the ventricles. Therefore it is essential that ventriculography be done in hospital, where a constant watch can be kept for evidence of the onset of hydrocephalus. This is the greatest risk in the procedure, but it can easily be avoided, if the possibility of its occurrence be kept in mind. The following point is worthy of emphasis. When a posterior fossa tumour associated with pronounced internal hydrocephalus is present, ventricular puncture results in a very sudden drop in the intraventricular pressure, which previously was very high; it is possible that the sudden relief of pressure may be followed by the occurrence of a multitude of minute hæmorrhages in the brain substance. It would be safer to deal with such conditions as we do with prostatic retention, that is, "decompress" by repeated small tappings of the ventricles, withdrawing only about twenty cubic centimetres at a time, and to administer some 50% glucose solution by intravenous injection. This would allow the brain to become adapted to the new pressure conditions. I have seen temporary collapse occur in two instances immediately following the complete reduction of pressure associated with pronounced hydrocephalus; the subsequent "cerebral irritation" might have been avoided by more gradual decompression. This gradual decompression would be particularly valuable as part of the preoperative treatment before suboccipital exploration in a case of severe internal hydrocephalus.

Ventriculography is a more risky procedure than encephalography, but occasionally there are tumours of which it is well nigh impossible to separate the signs; a parieto-occipito subcortical tumour may simulate a frontal or cerebellar lesion and *vice versa*. A ventriculogram will practically always settle the localization; this is its chief indication.

Some six years ago, while investigating patients suffering from post-traumatic headache, Wilder Penfield found that in a fair percentage of cases the subarachnoid injection of air caused, first, an increase of the headache and, finally, a relief which in some instances was permanent, in other temporary; in the latter more permanent relief was obtained by subsequent injections. Of the patients at the out-patient department on whom I have tried it, in only two could the results be classed as successful; but it is well worth while persevering with the method, as nothing else appears to give relief. It is believed that the headaches are due to the occlusion of the normal subarachnoid channels, especially in the frontal region, where they are most numerous; the injection of air separates the adhesions between the arachnoid and the cortex.

### Spinal Manometry.

A great deal of interesting information can be gained by using a manometer every time a lumbar puncture is performed. One such item of information resulted from doing routine spinal manometry in a series of cases labelled "post-traumatic

neurasthenia" (a term suitable enough providing it is used only as a label and is not meant to imply the absence of a pathological condition). As previously stated, the accepted limits of normal pressure are from 150 to 200 millimetres of water. In this series it was found constantly that the spinal fluid pressure were very low, the figures varying from 120 millimetres down to as little as 50 millimetres of water.

This fact was particularly striking in the case of a nurse who fell and struck the back of her head. There was no loss of consciousness, but on admission to the Adelaide Hospital she was slightly irritable, had pronounced photophobia, a pulse rate of 50 per minute and a respiratory rate of eight per minute; there were no other neurological signs. She remained in this state for some days. There was an occasional period in which she suffered from intense headache; at the same time there was a drop in the rate of pulse and respiration to 40 and six per minute respectively. All this suggested attacks of cerebral œdema, but there were still absolutely no other abnormal signs. Finally, during one of the attacks of apparent increase of intracranial tension, spinal puncture was performed with some apprehension; the fluid was quite clear and the maximum pressure was only 50 millimetres of water. Recovery commenced forthwith. A week after her discharge from hospital she was readmitted in the same condition, but she now lay in a position of opisthotonos, with her hands and fingers flexed in a typical hysterical posture. Spinal puncture was again performed; the pressure was still below 70 millimetres of water. About 30 cubic centimetres of fluid were replaced by air. Again she improved rapidly and is now back at her work.

I have not seen this point mentioned elsewhere, but it probably has been recorded; my series is far too small to be able to dogmatize thereon. But it does seem that the very low intraspinal pressure is sufficiently constant as to be of diagnostic value in functional cases simulating organic intracranial disease.

It is also of interest to note that the symptom of headache is associated with varying conditions of intracranial pressure. Cerebral œdema and mild hydrocephalus give rise to an intense headache, but the same type of headache is felt when the fluid is replaced by air at normal pressures, and, lastly, the headache associated with very low pressure readings is just as intense as in the conditions which produce an increase in pressure. Children with severe hydrocephalus and separation of the sutures frequently have little or no headache. During operations under local anæsthesia the only time when pain or headache is felt is when the meningeal or cerebral arteries are manipulated. Increase or decrease of intracranial pressure may, perhaps, cause headache by tension, or, on the other hand, by drag on the walls of the intracranial arteries.

The following case history illustrates the value of doing spinal manometry when there is persistent sciatica.

The patient, a male, aged fifty years, for seven years complained of severe pains in the thighs and down both legs, together with feelings of numbness and cold in the legs; the pains were increased by walking, standing, straining or coughing *et cetera*; he was unable to walk more than about a quarter of a mile without sitting down to rest, when the pain gradually eased off. X ray examination revealed widespread chronic arthritic changes in the dorso-lumbar vertebral articulations. He had no rectal or vesical disturbance, nor could any definite muscular involvement be detected. It is uncommon to find an obstructive cord lesion without any motor changes, but in spite of that, spinal manometry was performed. The first puncture was made between the fourth and fifth lumbar vertebræ; the fluid in the manometer then had none of the normal fluctuations. A second needle was inserted between the twelfth dorsal and first lumbar vertebræ. Observation of the fluid in the second manometer tube revealed normal respiratory and cardiac pulsations and normal rise and fall in response to compression of the jugular veins; in the lower or more caudal tube the response to jugular compression consisted of a very slow rise to 200 millimetres of water, then a very slow and incomplete fall to only 150 millimetres. This would be interpreted as evidence of a lesion causing partial or incomplete block. The colour of the fluid was normal, there was no increase of globulin nor of cells; the blood and fluid did not react to the Wassermann test. To confirm the manometry findings Dr. Nott injected "Lipiodol" into the *cisterna magna*. X ray examination revealed a partial and irregular obstruction to the passage of "Lipiodol" at the level of the body of the third lumbar vertebra. This was still present six days later. This is probably a case which French neurologists have recorded as chronic adhesive posterior arachnoiditis; the present intention is to perform a laminectomy and to endeavour to relieve the partial compression.

Time does not permit even a brief description of the outstanding feature of modern neuro-surgery, namely, the diagnostic and operative technique which has been evolved by Harvey Cushing and which has been adopted by every clinic which I visited. Cushing's gentleness in manipulation, his patience, his respect for the smallest bleeding point, his scrupulous care in the closure of wounds, permitting repeated incursions into the same tumour in safe stages without risk of infection, are some of the many points which impress the visitor to his clinic. It would seem that there is no short cut to success in neuro-surgery; the only hope of achieving anything comparable with Cushing's results and advances lies in trying to follow in minute detail the methods which are based on his enormous experience.

## OPHTHALMOLOGICAL ASPECTS OF PSYCHIATRY.[1]

By REG. S. ELLERY, M.D.,

*Neurologist to the Victorian Lunacy Department and Clinical Assistant to the Psychiatrist at the Melbourne Hospital.*

I COUNT it a privilege to have been asked to address this section, knowing that of all those present I am unquestionably the least qualified to speak on the subject of ophthalmology. For this reason I propose to avoid all reference to diseases of the eye as such, and confine my remarks to some of the ocular signs manifest in the diseases of the central nervous system.

The neurologist is justifiably proud of his knowledge of the nerve tracts and their connexions in the cerebro-spinal axis and of his ability to diagnose within the difficult domain of cortical localization. Manifestations of nervous disorders at the sensori-motor level so excite his interest that he frequently

[1] Read at a meeting of the Ophthalmological Section of the Victorian Branch of the British Medical Association on September 2, 1930.

overlooks or scorns the manifestations at the higher symbolic levels. This attitude is to a large extent responsible for the maintenance of the cleavage between neurology and psychiatry. It began, no doubt, at a time when disease was synonymous with demonology, when a palsy was the expression of divine punishment and a delusion the engendered insult of a devil. Echoes of this mediæval attitude of mind are still audible in modern medicine when lesions within the central nervous system become exteriorized in those conduct disorders ignominiously branded as "lunacy."

Now the central nervous system functions as a biological unit. Viewed simply, it consists of receptor neurones and effector neurones, between which are interposed a highly complicated set of internuncial neurones. Any of these neurones may be the subject of a lesion—traumatic, toxic or degenerative. According to our old conceptions, a lesion involving any part of the conducting mechanism would lead to certain disorders of function expressed variously in altered reflexes, anæsthesias and paralyses, while a lesion confined to the internuncial mechanism would cause disordered thinking and aberrant conduct—delusions and self-mutilations *et cetera*. The former constituted the province of neurology, the latter were lumped together as "lunacy" or more euphemistically referred to as the "problems of psychiatry." There is nothing commendable, either pathologically, clinically or therapeutically, in this divorce between neurology and psychiatry. In fact, it has been definitely disadvantageous, and I hope to show by a short, selective reference to certain ocular signs that they are as important to the psychiatrist as to the neurologist, because they are manifestations of lesions affecting an organ of complete biological unity.

### Neurosyphilis.

Consider some of the ocular manifestations of syphilis of the central nervous system. An analysis of 300 cases made by Joffroy[1] showed the percentages of ocular signs as set out in the following table:

*Percentages of Ocular Signs in Neuro-Syphilis.*

| Sign. | Percentage |
|---|---|
| Alteration of light reflex | 78 |
| Inequality of pupils | 68 |
| Abolition of light reflex (bilateral or unilateral) | 52 |
| Abolition of light reflex (bilateral) | 44 |
| Irregularity of pupil | 39 |
| Irregularity of both pupils | 36 |
| Diminution of light reflex | 36 |
| Diminution of light reflex (bilateral) | 26 |
| Alteration of accommodation reflex | 26 |
| Diminution of accommodation reflex | 17 |
| Mydriasis | 13 |
| Myosis | 13 |
| Diminution of light reflex (unilateral) | 11 |
| Abolition of accommodation reflex | 11 |
| Diminution of accommodation reflex (bilateral) | 9 |
| Abolition of accommodation reflex (bilateral) | 8 |
| Diminution of accommodation reflex (unilateral) | 7 |
| Fundus changes | 7 |
| Vascular changes | 5 |
| Abolition of accommodation reflex (unilateral) | 4 |
| Paresis of third nerves | 3 |
| Ptosis | 3 |
| Irregularity of one pupil | 3 |
| Nystagmus | 2 |
| Visual acuity lost | 2 |
| Atrophy of disc | 2 |
| Total blindness | 2 |
| Paralysis of fourth nerve | 1 |

This table may at first sight seem confusing, but it should appear clear if one remembers that these divers ocular changes are conditioned by the site of the lesion rather than by its nature. Neurosyphilis is in the nature of a toxi-infective process and is divided somewhat arbitrarily into types, tabetic, meningitic, paretic *et cetera*, according to the distribution through the nervous system of the lesions produced. The day has passed when one endeavoured to differentiate a hypothetical primary parenchymatous syphilis from the equally hypothetical meningo-vascular variety. But an alteration in pupillary response is so common and so extensive in all varieties of neuro-syphilis that such signs are looked upon by some as practically pathognomonic of the disease. This, however, is fallacious. In the statistics compiled by Bumke, in 36% of tabetics and 38% of paretics the Argyll-Robertson reaction was absent; and, as we now know, in many other diseases of the central nervous system there may be observed pupillary anomalies similar in every way to those elicited in neuro-syphilis.

I should like to say in passing that the pupillary reactions of neuro-syphilis which represent neuronic dissociation, are, in the earlier stages of the disease, irritative in origin, and consequently vary from time to time and not only as the result of treatment. Variation of pupillary inequality and the character of the light and accommodation reflexes has been frequently seen. A neuro-syphilitic lesion at this stage, that is, while the pupillary reactions are variable from time to time, frequently responds well to treatment either by malaria or other suitable form of fever therapy. However, when these signs have become static—presumably owing to actual neuronic destruction—the response to treatment is less good, although other symptoms due to involvement at other levels may be favourably influenced.

A number of such cases has come within my own experience.

### Disseminated Sclerosis.

Referring to disseminated sclerosis an analysis[2] of 141 cases showed that nystagmus was present in 70%, diplopia in 29%, pallor of the optic discs in 32%, transient ocular palsies in 29%, pupillary irregularities in 4·3% and the Argyll-Robertson reaction in 8·5%.

I do not propose to discuss the ætiology of disseminated sclerosis nor the pathology of its characteristic lesions, further than to say that recent work has advanced the claims of an infective causation and that the clinical picture, particularly the diversity of the ocular signs, would seem to endorse such claims. Like neuro-syphilis, disseminated sclerosis sets up reactions at the symbolic level, such as intellectual impairment, defects of orientation and retention, amnesia and fluctuations

of the affect which typify an "organic reaction syndrome"; one or other symptom probably predominates in every given case. It is only the fact that the initial symptoms of this disease are almost always confined to the neuronic conducting mechanisms and often those in the lower levels of the spinal axis that disseminated sclerosis has been relegated to the province of neurology; although, pathologically, the condition in the later stages presents evidence of a widespread tissue destruction entirely coextensive with the polymorphic nature of its clinical symptomatology.

### Epidemic Encephalitis.

The pathology of epidemic encephalitis is probably the most extensive, as the clinical signs are the most numerous and variable of all the infections of the central nervous system. The protean nature of its symptomatology and the seeming predominance of particular focal signs in different epidemics render the classification of symptoms extremely difficult. Practically every variety of ocular disturbance, including changes in the ocular fundi and the muscles of accommodation, and dissociation of the light reflex, has from time to time been reported, each one serving the more strongly to emphasize the toxi-infective nature of the disease and the necessity for the consideration of the nervous system as a whole in its reactions to this infection.

There can be no doubt that cases of epidemic encephalitis do occur in which the psychological component of the reaction has the most meagre mental colouring and in which changes in the ocular fundi of post-neuritic inflammatory origin with diplopia and ophthalmoplegia stand out so prominently in the clinical picture that reactions at other levels are overshadowed. On the other hand, I have seen committed to mental hospitals not a few patients whose rapidly changing delusional ideas and psychomotor restlessness, or disorientation with somnolence almost of the catatonic type, have, by reason of their dramatic character, taken precedence over neurological signs. Thus these patients have died, for their illness had remained undiagnosed and untreated. Cases hitherto classed as "acute confusional insanity" or "acute delirious mania" have at times been found at *post mortem* examination to have been encephalitic in origin. It is in such cases, I think, that the separation of neurology from psychiatry, previously referred to, has been so detrimental. For a knowledge of the focal phenomenology of acute infective conditions of the central nervous system, assisted by the knowledge gained from an ophthalmoscopic examination, and a knowledge on the part of the neurologist of the psychological reactions to infection, would prove to the best advantage of the patients, both for the purposes of diagnosis and therapeusis.

### Other Toxi-Infective Conditions.

The point just made is further emphasized in the case of toxæmias involving the central nervous system. Alcohol, for example, has a widespread action on the central nervous system, but, speaking generally, the neurologist deals only with alcoholic neuritis, relegating such chronic forms as alcoholic hallucinosis and other clinical varieties to the psychiatrist. But pathologically the effects of alcohol are by no means confined to peripheral nerves or effector neurones. Alcohol, acting as a toxin, involves the nervous system as a whole with a tendency to focal emphasis for which there is at present no adequate explanation. Among focal symptoms pupillary irregularities are common. Menninger[3] in an analysis of 58 cases of alcoholism found pupillary inequality in 13·8% and abolition of the light reflex in 36·2%. Kinnier Wilson[4] mentions a patient in whom the typical Argyll-Robertson reaction could be elicited; ocular palsies and nystagmus have been reported as occurring in association with the typical compensatory confabulation and peripheral neuritis of the Korsakow syndrome. In fact, so closely in some cases of alcoholism do such symptoms as ataxia, dysarthria and tremor combined with an expansive delirium and an Argyll-Robertson pupil mimic the classical syndrome of "general paralysis" that clinical differentiation may tax the skill of even the most expert investigator.

Not to labour the point, I shall merely state, without qualification, that ocular signs, while in no sense pathognomonic, may be elicited in other toxic states, such, for example, as in diabetic coma, uræmic coma and carbon monoxide poisoning, the concomitant psychological symptoms being those of toxic deliria generally.

### Meningeal Lesions.

When there are severe meningeal hæmorrhages of whatever origin the pupils vary greatly. In fact, they are so variable that they are of very slight diagnostic value. Changes in the ocular fundi also occur, but they merely serve to indicate the degree of intracranial pressure.

Inflammatory lesions of the dura arising secondarily from purulent conditions of the frontal sinuses or middle ear or (with greater frequency in the insane) from erysipelas, give rise to generalized signs of cerebral irritability. *Pachymeningitis hæmorrhagica interna* may produce a number of psychotic symptoms. It is a condition associated with advancing years and, apart from neuro-syphilis, it is often the terminal state of a chronic mania. Headache, nausea and confusion which is frequently of an irregular, intermittent type, may be the initial symptoms. According to the site and extent of the lesion various ocular signs may be seen. Conjugate deviation and nystagmus have been noted, but some grade of papillitis is more often present, accompanied by myotic or irregular pupils and depression of the light reflex. The psychological symptoms may develop along the lines of a senile psychosis with a gradually appearing amnesia and impairment of retention, childish irritability, defects of orientation and a general decline of mental capacity. Or a patient far advanced in the

dementia of senility may, as the result of some apparently trifling trauma, develop an acute pachymeningitis with rapid extravasation, producing epileptiform convulsions, myoclonic twitchings, hemiparesis, aphasia or coma in the successive wake of increasing pressure symptoms. Ocular signs, taken in conjunction with these in such a case, should leave but little room for a doubtful diagnosis in the mind of the psychiatrist.

Acute inflammatory conditions of the pia-arachnoid of varied ætiology may give rise to any of a large number of ocular signs ranging from mild ptosis to convergent strabismus and diplopia. Papillitis may be prominent, but it is not of itself diagnostic. Mental symptoms make an early incursion into the clinical picture and are usually those of a mild toxic delirium and often considerable motor restlessness.

Occurring secondarily to neuro-syphilitic infections and in cases of senile dementia, the signs and symptoms produced by chronic leptomeningitis in the majority of instances cannot be clinically distinguished from those engendered by the primary lesion. Chronic leptomeningitis is frequently observed *post mortem* in the chronic insane; it may give rise to symptoms of intracranial pressure with the development of internal hydrocephalus by the formation of adhesions tending to the obliteration of the subarachnoid space and impeding the normal circulation and exit of the cerebro-spinal fluid. Pupillary signs and disc changes are all too seldom sought, but chronic leptomeningitis may be the cause of the pupillary anomalies not infrequently observed among the chronic insane.

### Cerebral Abscess.

While cerebral abscess may present a major problem to the surgeon, both in the matter of diagnosis and in localization, it plays a relatively minor rôle in the ætiology of the psychoses. It was present on two occasions only in a series of 193 consecutive *post mortem* examinations which I recently conducted on the bodies of insane persons. I mention it here only because the ocular changes which may accompany cerebral abscess may assist in the diagnosis and the differentiation of specific symptoms from those of biogenetic origin.

Psychiatrically speaking, a cerebral abscess may be primary or secondary; that is, psychological symptoms may be caused entirely by the abscess or an abscess, frequently of metastatic origin, may develop in a psychotic patient and thereby engender a set of physical signs with consequential modification of the clinical picture.

In the first type of case, when the abscess most often is the result of spread from *otitis media*, the "mental" symptoms may mislead the physician and the patient be certified and sent to the receiving house. The following brief records illustrate this contention:

E.C., a woman aged nineteen years, following a miscarriage, developed auditory hallucinations, hearing the voice of God. She became confused and apprehensive, and in consequence she was certified and admitted to a receiving house. Her temperature was 37·8° C. (100° F.). Four days later she developed paresis of the left arm; three days later again she had a convulsive seizure without loss of consciousness, but with twitchings on the left side of the face, the left arm and leg. The following day she complained of frontal headache. Her pulse rate was 110 per minute and her temperature was 37·8° C. (100° F.). A week later she was completely paralysed down the left side; she was vomiting; her consciousness was partially clouded. Vomiting and headache continued intermittently for a fortnight. The paralysis remained unchanged. She was confused and restless and died exactly one month after admission. *Post mortem* examination revealed chronic myocarditis with some recent bronchitis and an abscess of the right cerebral hemisphere in the motor area.

A.S., a single man aged forty-three years, was admitted to the receiving house. Certificates stated that he was dull and apathetic and without interest in his surroundings, that he was disorientated and complained of headache. His reflexes were found to be generally exaggerated; his pupils were small and unequal; the light reflex was absent and the accommodation reflex was sluggish. No ophthalmoscopic examination was made. A fortnight after admission he was reported to be dull and resistive, unable to give any information about himself, although his perception appeared normal. Clouding of consciousness ensued and his habits became defective. He died five days later. *Post mortem* examination revealed hypostatic congestion of the lungs and the brain which was under pressure from an abscess of the right temporo-sphenoidal lobe (the so-called symptomless abscess).

A.H.S., a man aged twenty-two years, was admitted to the receiving house with symptoms of confusional insanity following a right mastoid operation performed in the country. At the time of admission he was dull and apathetic, slow of thought and lacking in objective interest. His memory was poor; he had vague persecutory delusions; his habits were defective. Physical examination revealed a normal reflex response everywhere save that the left superficial abdominal reflexes were absent. The right pupil was larger than the left, but both responded normally to light and accommodation. Ophthalmoscopic examination was not then performed. Neither blood nor cerebro-spinal fluid reacted to the Wassermann test. He remained in this anergic state for two months, when he was examined by an oculist who reported "choroiditis with pale discs very suggestive of congenital syphilis." A month later he commenced vomiting. His general weakness increased and his consciousness became clouded. For some days he remained lethargic with occasional outbursts of restlessness in which he appeared to be in pain, moaning and putting his hands to his head; then quite suddenly he died. *Post mortem* examination revealed some old pleural adhesions of the right lung and some tubercular scarring at the apex. The meningeal vessels were congested and the convolutions were flattened as by pressure. Cerebro-spinal fluid escaped under great pressure and a large abscess in the right cerebellar lobe ruptured as the brain was being removed.

No other diagnosis but "confusional insanity" was made in these cases; as it is not mentioned in the clinical notes cerebral abscess was apparently not suspected.

In the second type of case in which cerebral abscess may occur, pronounced alteration in the symptoms of a patient suffering from long-standing mental disorder may perplex the psychiatrist unless the physical signs are correctly assessed in conjunction with the finding of a primary focus. Ocular changes are frequently prominent in such cases, their character depending upon the site, size and age of the abscess, but obviously they are not of themselves diagnostic.

W.J., a man aged twenty-nine years, was admitted to a mental hospital suffering from systematized delusional insanity. At the time of admission he was reported to have been in good bodily health. He was exalted in manner and speech by reason of his delusions of inherited wealth. Twenty years went by without much change in his mental condition. His delusions suddenly became less apparent. He was emotionally torpid and his attention could not be held. He became drowsy and stupid. His reflexes were exaggerated. His pupils were unequal and failed to react to light. The ocular fundi were not examined. He remained in this state for some weeks, when vomiting commenced. His temperature is not recorded, but the vomiting continued and within two days he died. *Post mortem* examination revealed some active tubercular foci in the left pulmonary apex. The brain surface was flattened; a large abscess in the left parietal lobe had ruptured into the lateral ventricle so that all ventricles contained pus.

This lesion adequately accounts for the sudden change in the personality and behaviour of a man who for twenty years had retained the same delusional system. The nature of the cerebral lesion was unsuspected, but might have been guessed at had the eye grounds been examined and the temperature recorded.

### Cerebral Neoplasm.

To review the extensive and varied semeiology of cerebral neoplasms which may result from a combination of tissue destruction and alteration of intracranial pressure would in this instance be valueless unless it could be correlated with specific psychological reactions. While it is established that tumour formation within the cranium is distinctly rare as a primary cause of a psychosis, many so-called "mental" symptoms arise as expressions of neoplastic insult in practically all cases of cerebral tumour. The majority of these, in the present state of our knowledge, we are unable to localize and thus frequently we are led into the realm of clinical contentiousness.

A tumour which in the beginning causes major involvement of the sensori-motor projection systems, will generally give rise to a clinical picture dramatic enough for accurate diagnosis. But when the initial growth is confined to cortical associational areas causing reactionary symptoms in the sphere of intellect and conduct, the nature of the lesion is most liable to be missed under the psychotic disguise. Arterio-sclerotic changes and the symptoms of intellectual deterioration generally which presage the onset of the involutional psychoses—defects of memory and judgement, impairment of insight and attention—are those which most commonly cloak the presence of a cerebral tumour. Ophthalmological examination in such cases may be the only means whereby the correct nature of the lesion is discovered.

It seems to me presumptuous on the part of some writers to endeavour to give a localizing value to such symptoms as, for example, hallucinations, agnosia, and disorientation. It may be that euphoric states and a tendency to "misplaced jocularity" accompany tumours of the frontal lobes, it may be also true that visual and auditory hallucinations become prominent in association with tumours of the parieto-occipital and temporal regions, but it would be dangerous indeed to regard any of these symptoms as indicative of specific lesions, as illustrated by the following histories:

B.C., a male aged twenty-eight years, was admitted to a mental hospital presenting the syndrome of *dementia præcox*. He had emotional obtundity and seemed withdrawn from reality. His attention could not be held and his mood changed at times from dreaminess to irritability. He had auditory hallucinations. Later he had generalized convulsions and developed the delusion (?) that his eyesight was failing. Death occurred during a convulsion and autopsy revealed a large, widely infiltrating growth of the right frontal lobe. His ocular fundi were not examined prior to his death.

D.J., a male aged thirty-four years, was admitted to a mental hospital from a psychiatric clinic, suffering from intense occipital headache and symptoms of confusional insanity. He was confused, disorientated and had hallucinations. Ophthalmoscopic examination revealed acute papillœdema. Vomiting commenced shortly after admission and a tentative diagnosis of cerebral tumour was made. He died shortly afterwards. The *post mortem* examination revealed an acute cerebritis, but no growth. This was probably a case of post-influenzal psychosis with choked discs resembling a cerebral neoplasm.

Our knowledge of the cerebral cortex and its functions is enlarging rapidly, but we are not yet justified, I think, in laying down any set diagnostic rules for the exact localizing significance of the specific psychological disorders. Headache, nausea and vomiting combined with changes in the optic discs may form a definite syndrome; but when either of these exists in combination with a variety of psychotic symptoms, it were better, I believe, to fall back and rely upon the results of ophthalmoscopic examination, lumbar puncture and encephalography than to theorize about the site and nature of a lesion from the often misleading premises of psychotic reactions.

### Summary and Conclusion.

I have, I confess, done little more than touch the fringe of this subject, for ocular signs bulk largely in the many disorders which come within the purview of the psychiatrist and the neurologist. There is much more that one could profitably discuss in relation to pupillary changes arising from cerebral trauma not due to syphilis or other infections. There are the ocular signs and psychological symptoms presented in the heredo-degenerations. There are the conjugate visual paralyses seen in certain drug intoxications associated with clouding of consciousness, disorientation and psychomotor unrest. There is the question of narcolepsy which may be associated with tumours of the pituitary region, causing ocular signs. There are retinal changes observed in presbyophrenia and in the organic dementias of the arterio-sclerotic type. There is that rare condition of epiloia, tuberose sclerosis with multiple gliomata of the retina, seen in the mentally deficient. There is that fascinating phenomenon known as the "oculo-gyric crisis" observed most frequently in the guise of a compulsion neurosis in post-encephalitic Parkinsonism, but occurring also with other psychotic manifestations in hysteria and sometimes in *dementia præcox*.

There are problems relating to visual hallucinations generally and specifically to the Lilliputian hallucinations and the eye as an erotic symbol. But the number is legion.

I would conclude this brief and all too inadequate survey by again pointing out:

1. That the further one goes in the study of the diseases of the nervous system the narrower seems the division between neurology and psychiatry; and that from the ophthalmological point of view such division seems to serve no useful purpose.

2. That the nervous system, being a biological unit, reacts as a whole to any lesion by which it may be affected, certain focal signs varying with the site and nature of the lesion, just as individual psychological symptoms depend upon constitution and personality.

3. That the ocular apparatus, for its complete integration and function, involves a number of complicated neuronic pathways and intercalated reflex arcs with extensive topographical relations; so that with regard to toxi-infective conditions within the central nervous system a multiplicity of ocular signs may become manifest, none of which is pathognomonic of any particular infection or toxin.

4. That some ocular signs are common to certain well recognized diseases of the nervous system; this does not invalidate their significance nor in any way abolish the necessity for thorough ophthalmological examination in all diseases of nervous origin, if only as an ancillary factor in differential diagnosis.

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## THE EXCRETION OF LEAD IN THE URINE AFTER INJECTION OF COLLOIDAL LEAD ORTHOPHOSPHATE.[1]


By ROLLO K. NEWMAN, B.Sc., A.A.C.I.

(*From the Physiological Laboratory of the University of Sydney.*)


IN some previous work[1] the author found that patients receiving relatively infrequent injections of colloidal lead excreted the metal in the urine in approximately the same concentration as was known to be found in cases of lead poisoning. Millet,[2] on the other hand, found, on investigating the urine of patients receiving injections of colloidal lead orthophosphate, that there appeared to be no excretion of lead in the urine.

To check these two findings the author, using a horse for the purposes of experiment, investigated the rate of disappearance of lead from the blood and the concentration of lead in the urine following an injection of each of these preparations.[3] It was found that lead disappeared more rapidly from the blood when injected as colloidal orthophosphate than was the case when the colloidal metal was injected. After injection of colloidal lead orthophosphate the concentration of lead in the urine gradually rose until, eighty minutes after injection, it amounted to 1·1 milligrammes per litre, whereas after injection of the colloidal metal preparation the concentration rose to 54 milligrammes per litre in the first thirty minutes and fell to 35 milligrammes per litre at the end of eight hours, thereafter continuing to fall steadily until at the end of a week it had reached 7·6 milligrammes per litre.

Unfortunately no samples were obtained after the first eight hours following the injection of phosphate and it is not known therefore what was the maximum concentration reached.

### Excretion of Lead in the Urine of a Patient.

Recently an opportunity occurred for an examination of the excretion of lead in the urine of a patient receiving a course of intravenous injections of colloidal lead orthophosphate in the Royal Prince Alfred Hospital.

The patient was a male, aged fifty-seven years, suffering from an epithelioma of the tongue and large glands in the neck. He was receiving no other treatment. The lead was administered as shown in Table I.

TABLE I.
*Injections of Lead Orthophosphate.*

| Date. | Lead Orthophosphate injected (in Milligrammes of Lead). |
|---|---|
| 25 .9.30 | 32 |
| 1.10.30 | 40 |
| 4.10.30 | 50 |
| 8.10.30 | 60 |
| 14.10.30 | 80 |
| 17.10.30 | 80 |
| 20.10.30 | 80 |
| 22.10.30 | 80 |
| 24.10.30 | 76 |
| 27.10.30 | 80 |
| 29.10.30 | 92 |
| 31.10.30 | 76 |
| 3.11.30 | 80 |
| 5.11.30 | 76 |
| TOTAL | 982 |

The clinical history shows that there were no toxic symptoms following injection at any time. On October 7, 1930, three days after the third injection, the patient complained of some pain in the tongue and left side of the neck, which lasted for a few days. After the eighth injection on October 22, 1930, similar pain was experienced. The patient was at this time improving in general condition and had gained 0·9 kilogram (two pounds) in weight. After the tenth injection, on October 27, 1930, there was some salivation which lasted for just over one week.

When discharged, the patient was greatly improved in general condition and the glands were harder and smaller.

Unfortunately the estimations of lead in the urine were not commenced until October 10, fifteen days after the beginning of treatment and two days after the fourth injection. From October 10 until November 2 the entire urine voided during the

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

TABLE II.

*Lead in the Urine After Injections of Colloidal Lead Orthophosphate.*

| Date (24 hours commencing 11 a.m.). | Injections (Milligrammes of Lead). | Total Lead Injected (Milligrammes). | Urine Excreted (Cubic Centimetres). | Total Lead in Urine (Milligrammes). | Concentration of Lead in Urine (Milligrammes per Litre). |
|---|---|---|---|---|---|
| September 25 .. .. .. | 32 | | | | |
| October 1 .. .. .. | 40 | 72 | | | |
| „ 4 .. .. .. | 50 | 122 | | | |
| „ 8 .. .. .. | 60 | 182 | | | |
| „ 10 .. .. .. | | | 900 | 0·180 | 0·20 |
| „ 11 .. .. .. | | | 1,250 | 0·188 | 0·15 |
| „ 12 .. .. .. | | | 1,650 | 0·247 | 0·15 |
| „ 13 .. .. .. | | | 1,250 | 0·238 | 0·19 |
| „ 14 .. .. .. | 80 | 262 | 1,520 | 0·335 | 0·22 |
| „ 15 .. .. .. | | | 1,050 | 0·199 | 0·19 |
| „ 16 .. .. .. | | | 735 | 0·176 | 0·24 |
| „ 17 .. .. .. | 80 | 342 | 1,200 | 0·226 | 0·19 |
| „ 18 .. .. .. | | | — | — | — |
| „ 19 .. .. .. | | | 825 | 0·155 | 0·19 |
| „ 20 .. .. .. | 80 | 422 | 1,175 | 0·223 | 0·19 |
| „ 21 .. .. .. | | | 1,190 | 0·238 | 0·20 |
| „ 22 .. .. .. | 80 | 502 | — | — | — |
| „ 23 .. .. .. | | | 1,500 | 0·285 | 0·19 |
| „ 24 .. .. .. | 76 | 578 | 1,530 | 0·306 | 0·20 |
| „ 25 .. .. .. | | | 950 | 0·275 | 0·29 |
| „ 26 .. .. .. | | | — | — | — |
| „ 27 .. .. .. | 80 | 658 | — | — | — |
| „ 28 .. .. .. | | | 1,470 | 0·294 | 0·20 |
| „ 29 .. .. .. | 92 | 750 | 1,605 | 0·321 | 0·20 |
| „ 30 .. .. .. | | | — | — | — |
| „ 31 .. .. .. | 76 | 826 | — | — | — |
| November 1 .. .. .. | | | 1,415 | 0·466 | 0·33 |
| „ 3 .. .. .. | 80 | 906 | — | — | — |
| „ 5 .. .. .. | 76 | 982 | — | — | — |

previous twenty-four hours was forwarded daily to the laboratory for analysis on all except six days. The method of estimation employed was that used previously for the estimation of lead in the urine of patients receiving colloidal lead injections. The results of the estimations are set out in Table II.

The figures in Table II show that in this case at least, during the period of observation, lead was undoubtedly excreted in the urine following the injection of colloidal lead orthophosphate. This would appear to be in direct contradiction to the findings of Millet, but it is possible that the difference may be explained as follows:

In Millet's cases the samples of urine were taken in three instances on the day of injection, in two instances on the day following injection, in one instance 12 days after injection, in a number of instances from 17 to 801 days after injection. The customary dose was 50 milligrammes of lead as phosphate. It seems possible, from examination of Millet's results, that the patients from whom samples were taken at a relatively short period after injection may have received only a single injection of 50 milligrammes of lead; when one takes into account the relative rate of excretion of lead after a single injection as the phosphate and as colloidal metal into a horse, it appears probable that the increase in the amount of lead in human urine following a single small injection of the phosphate would be scarcely appreciable. In the other instances in which more lead may have been administered, it is possible that the rate of excretion had greatly fallen off before samples were taken. No samples were taken between one and twelve days after injection, and the majority were taken at longer periods than a month after injection.

The results obtained in the present instance, when arranged as in Table III, show that the actual amount of lead excreted on the days of injection was greater than on intermediate days; but this was due to an increase in the quantity of urine excreted and not to an increase in the concentration of lead in the urine. This is in marked contrast to the effect of colloidal lead injections, following which there is usually a definite fall in the urine excretion and high concentration of lead.

It is an important distinction between lead orthophosphate and colloidal lead that injections of the former do not cause great immediate increase in the concentration of lead in the urine, whereas injections of the latter may result in a concentration of lead in the urine averaging 0·21 milligramme per litre for the week following the first injection and for the weeks following the second and third injections, 0·70 to 0·90 milligramme per litre.

In the previous work on excretion of lead in the urine the average weekly figures were considered rather than the daily figures. Although on several

TABLE III.

*Comparison of Average Daily Excretion of Lead on Days of Injection and Intermediate Days.*

| | Average Volume of Urine (Cubic Centimetres). | Average Amount of Lead Excreted (Milligrammes). | Concentration of Lead in Urine (Milligrammes per Litre). |
|---|---|---|---|
| Days of injection (5) .. .. .. .. .. | 1,406 | 0·282 | 0·20 |
| Intermediate days (12) .. .. .. .. .. | 1,180 | 0·245 | 0·21 |

TABLE IV.
*Average Weekly Excretion of Lead.*

| Period. | Lead Injected during Period (in Milligrammes). | Total Lead Injected (in Milligrammes). | Lead Excreted during Period (in Milligrammes). | Average Concentration of Lead in Urine (in Milligrammes per Litre). |
|---|---|---|---|---|
| October 10–16 .. .. .. .. | 80 | 262 | 1·56 | 0·187 |
| October 17–23 .. .. .. .. | 240 | 502 | 1·61 | 0·192 |
| October 24–30 .. .. .. .. | 248 | 750 | 1·80 | 0·215 |
| November 1 (one day only) .. .. | | 826 | 0·466 | 0·330 |

days the whole of the urine voided in the preceding twenty-four hours was not available, it is possible to calculate from the specimens obtained the average weekly excretion over the period of the investigation, assuming a fairly constant weekly urine output.

The figures thus arranged in Table IV show that frequency of injection does not have much bearing on the output, since in the first week dealt with, when only 80 milligrammes of lead were injected, the output was similar to that for the second week, when 240 milligrammes were injected. But there is some slight indication of a tendency to increased lead output and increased concentration in the urine as treatment progresses.

### Comparison of the Excretion of Colloidal Lead and Colloidal Lead Orthophosphate.

On account of the great disparity in the amounts of lead injected, it is somewhat difficult to draw a comparison between the figures in Table IV and the figures obtained previously for the lead excretion of patients receiving treatment by means of injections of colloidal lead. One patient, for example, received only 160 milligrammes of colloidal lead during fourteen weeks of treatment, another 158 milligrammes in eleven weeks, another 112 milligrammes in six weeks; the average concentration of lead in the urine for the entire period in these cases was 0·38, 0·42 and 0·32 milligramme per litre respectively. What the lead concentration in the urine would reach if 982 milligrammes of colloidal lead were injected in six weeks, it is difficult to imagine, since, as has been stated, concentrations of 0·7 to 0·9 milligramme per litre may be maintained for a week after two injections of 70 milligrammes of colloidal lead given at an interval of longer than one week.

### Conclusion.

The results of this work appear to confirm the observations previously made regarding the difference between the behaviour after injection of colloidal lead and colloidal lead orthophosphate. It would appear that after injection lead orthophosphate is not excreted as such nor is it quickly changed in the blood into any excretable form, hence an injection produces no immediate increase in the concentration of lead in the urine. The lead orthophosphate is, however, rapidly filtered out of the blood by various tissues and when once these tissues have absorbed more than a certain minimum amount, they commence to reliberate lead into the blood in some form in which it can be excreted. The rate at which the tissues liberate this excretable lead compound is fairly constant and only increases very slowly as successive injections increase the amount of lead contained in the body.

On the other hand, the process of elimination of colloidal lead seems to differ in the following way. After injection, although there is still a filtering out by the tissues, a considerable amount of the lead is either directly excreted or quickly converted in the blood into an excretable form, so that there is an immediate rise in the lead concentration in the urine. The concentration gradually falls, however, to a certain level, approximately of the same order as that brought about by injections of lead orthophosphate. During this period the lead originally absorbed by the tissues is probably reliberated into the blood in a similar form and at approximately the same rate as occurs when lead is injected as phosphate.

### Acknowledgement.

The author would like to acknowledge his indebtedness to Dr. R. H. Kenny, of the Royal Prince Alfred Hospital, who suggested that this work be carried out upon his patient and arranged for the collection and forwarding of samples to the laboratory.

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## THE TREATMENT OF DEAFNESS BY THE ZÜND-BURGUET ELECTRO-PHONÖIDE METHOD: SUMMARY OF FORTY CONSECUTIVE CASES.[1]

By R. GRAHAM BROWN, F.C.S.A.,
*Brisbane.*

SET out in tabular form below is a rough summary of results obtained in the treatment by means of the Zünd-Burguet method of forty patients suffering

[1] Written in response to a request for information in Dr. R. H. Bettington's article on "The Treatment of Deafness by the Zünd-Burguet Method," THE MEDICAL JOURNAL OF AUSTRALIA, September 13, 1930, page 366.

from deafness. These patients were the first who underwent this treatment at my hands.

As I believe that I was the first aurist in Australia to make use of the method in the treatment of deafness (being closely followed by Dr. Blashki, of Sydney), I feel it my duty to publish my experiences.

The method had previously interested me for some years, and during the early part of 1926 I visited Paris especially in order to obtain first hand information of Zünd-Burguet's apparatus and his method of treatment. Zünd-Burguet is a scientific man, but not a practical otologist. In fact, he is not medically trained, and I soon discovered that he was not even competent to sort out the varieties of deafness. At the same time, in spite of these adverse factors, I was sufficiently impressed by what the patients seen by me (all of whom, by the way, happened to be women) said concerning the results of the treatment, that I invested in one of his machines, for I considered that even if it were possible to obtain one-tenth of the good results that Zünd-Burguet claimed for his method, it would be worth a patient's while to undergo the course of treatment. I started off enthusiastically, but after carefully observing the forty consecutive patients detailed here, the results obtained satisfied me that the method was far from scientific as regards the treatment of deafness and noises in the head, and the benefits derived were very few and slight. In deaf people, as has been remarked before, we have not only the deafness and noises in the head to contend with, but also that "fullness in the head" which is a frequent symptom. In regard to this last factor, most of my female patients suffering in this way were satisfied that they were improved by the treatment, but not so the male. Noises, in only a few cases, were lessened to a degree which made the treatment worth while.

Of the 40 patients detailed, 26 were females and 14 males. It is interesting to note that only three of the 14 males improved in hearing and this improvement was only of a very transient and questionable nature. This brings out a characteristic which is frequently observed in deaf females, that is, they are very easily "buoyed up" by almost any form of treatment and give responses to tests which must be carefully scrutinized; otherwise the practitioner may be misled into believing that they hear better than they do. Males are very much less liable to this fault. Amongst the females in the series the best result obtained was in Case 9, a doctor's wife. She was definitely improved, but retrogressed in a short period after each course (three in all). Subsequently she improved greatly after Wood's intratympanic iodine treatment, but subsequently relapsed, only to improve with further treatment. Patient 11 was a highly-strung music teacher. She is convinced that the treatment cleared her head of fullness and noises. Patient 13 is worth mentioning as one of a class of people who become obsessed with treatment and its imagined beneficial effects. I still had difficulty after completing several courses in making her understand what was said by using a "loud voice." No improvement was apparent to tests and she still complained that "her noises were sending her mad."

I feel that the public should not be buoyed up with hopes regarding the results to be expected from this form of treatment. Very early in my experience I realized this and made it a practice to inform my patients definitely before beginning treatment that the benefits derived would at most be but small. As in all other instances of certain lines of treatment, we have our enthusiasts among the practitioners and among the patients, and in a few instances patients have worried me to continue the treatment, in spite of my being convinced that it was not doing them any good.

I have tried this method on four deaf mutes and can affirm that no improvement whatsoever resulted. When one considers that to carry out Zünd-Burguet's directions in their entirety entails the application of his vibration instrument (which, by the way, is an ingenious electrical one) to the mastoid region, the back of the neck, down the spine and even to the abdomen, and that Zünd-Burguet insists upon the supplying of this molecular vibrator as part of his outfit, one realizes that there is a certain amount of quackery about the method. The treatment entails anything up to 80 *séances*, each lasting eight minutes, and it is obvious that these take up a lot of the specialist's time. I have relegated this treatment, which I seldom use now, to my nurse, who has learned to do it as efficiently as myself, and only a very nominal fee is charged. That a practitioner should charge, as is done by certain specialists in England and elsewhere, full fees for this form of treatment is, I think, not justified by the results obtained.

In conclusion, I consider that the summary of my first forty cases has emphasized that at most but little good results from this form of treatment. I have been persuaded to record my experiences in order to support this conclusion, and also to warn the general practitioner and through him the public, that the claims of Zünd-Burguet and his followers should be received with caution. I think that the method belongs even to a lower class than the treatment of deafness by galvanism, pneumatic massage, ultra-violet rays and that popular form of treatment, it seems for all ailments, diathermy. In the treatment of deafness we can generally help our patients, sometimes to a surprising degree, by undertaking treatment along the various accepted avenues, namely, by directly improving the patency and condition of the Eustachian tubes and middle ears; by rectifying nasal and naso-pharyngeal conditions, which indirectly improve the aforementioned structures; by making injections into the middle ear itself through the drum, after the method of Wood; by Vodak's (of Prague) treatment by sodium arsenate given internally; by the internal administration of small amounts of iodine over long periods; by the judicious use of extracts of the endocrine glands, which probably act directly on the acoustic nerve, *et cetera*.

SUMMARY OF RESULTS OF ZÜND-BURGUET'S ELECTRO-PHONÖIDE TREATMENT OF DEAFNESS.

| Number. | Sex. | Age. | Deafness. | Noises. | Fulness. | Result. |
|---|---|---|---|---|---|---|
| 1 | F. | 64 | | Continual. | + | Discontinued after five *séances*. "Noises made worse." |
| 2 | F. | 35 | Air conductive, right nerve. | Right + | | Slight improvement in hearing. (?) Due to previous other forms of treatment. |
| 3 | F. | 29 | Air conductive, paracusis. | Perpetual six months. | + four years. | Clinically there is no improvement in hearing. |
| 4 | F. | 63 | Air conductive. | + | | No difference after forty-three *séances*. |
| 5 | F. | 62 | Air conductive, slight nerve. | Nil now. | | No difference. |
| 6 | F. | 24 | Air conductive, paracusis. | + | | No improvement. |
| 7 | F. | 44 | Air conductive. | + | | No improvement. |
| 8 | F. | 21 | Air conductive, paracusis. | + | + | Unsatisfactory type, gave up treatment. |
| 9 | F. | 24 | Air conductive. | + | + | Improved in every respect, but retrogressed; had three courses in all. |
| 10 | F. | 36 | Air conductive, slight nerve, paracusis. | | | After eighty *séances* no improvement at all. |
| 11[1] | F. | 28 | | + | + | She states there is a very great improvement. |
| 12 | F. | 58 | Air conductive, slight nerve. | | | After 101 *séances* she states she is better in health and her hearing has improved considerably. Clinically there is no improvement. |
| 13[2] | F. | 60 | Air conductive. | ++ | | Patient stated in two weeks she could hear better; "Heard alarm clock ticking and also wristlet watch." |
| 14 | F. | 65 | Air conductive, nerve. | | | Sometimes thinks she is better. |
| 15 | F. | 30 | | + for months. | | Disappeared after three *séances*. |
| 16 | F. | 36 | Air conductive, paracusis. | + | | During treatment patient was very pleased and said she was improved in hearing and that noises were less. Later reported previous condition returned, but it was no more than before treatment. |
| 17 | F. | 58 | Air conductive, slight nerve. | + | | No difference. |
| 18 | F. | 33 | Air conductive, slight nerve. | ++ | | Thinks she can hear better, but tests reveal no improvement. |
| 19 | F. | 44 | Air conductive. | + | + | No difference. |
| 20 | F. | 48 | Air conductive, nerve, and paracusis. | + | | No difference after forty-five *séances*; left off treatment. |
| 21 | F. | 27 | Air conductive, paracusis. | + | + | Improved for a short time and then returned to previous condition. |
| 22 | F. | 28 | Early air conductive. | + | | Four separate short courses. Had to stop each on account of noises which were made worse by treatment. |
| 23 | F. | 59 | Advanced air conductive. | + | | No improvement hearing or relief from noises. Better in general health and sleeps better. |
| 24 | F. | 64 | Advanced conductive and slight nerve. | ++ | | After first course, said she heard better; after second course, no improvement. |
| 25 | F. | 40 | Conductive + | + | | After forty-two *séances*, hearing better. Tests revealed no change. Later she said she was no better than before treatment. |
| 26 | F. | 72 | Nerve ++ | + roaring. | + | Says "hearing improved wonderfully," and her friends say she hears better. No difference in tests. |
| 27 | M. | 35 | Air conductive. | + | + | No improvement. |
| 28 | M. | 7 | Deaf mute. | | | No improvement. |
| 29 | M. | 59 | Very advanced nerve. | | | No improvement to tests, but says he can hear the trams better. |
| 30 | M. | 27 | Air conductive, paracusis. | ++ | + | He says he improved temporarily, but previous condition returned. |
| 31 | M. | 14 | Air conductive, paracusis. | | | No improvement. |
| 32 | M. | 11 | Partial deaf mute. | | | No difference in hearing. |
| 33 | M. | 9 | Air conductive. | | | No improvement. |
| 34 | M. | 16 | Advanced air conductive, early nerve, paracusis. | | | "No better in hearing, but better in health." |
| 35 | M. | 35 | Advanced air conductive. | + | + | No improvement. |
| 36 | M. | 35 | Air conductive, slight nerve. | + | | "No improvement in hearing, but noises have gone." |
| 37 | M. | 55 | Air conductive, paracusis. | ++ | | Slight improvement in hearing and relief from noises while under treatment. Return to former state. |
| 38 | M. | 47 | Nerve. | + | + | No improvement. |
| 39 | M. | 38 | Air conductive. | + | + | No improvement. |
| 40 | M. | 16 | Air conductive. | + | + | No improvement. |

[1] This patient was a music teacher whose ears were very sensitive.
[2] This patient haunted my surgery and demanded continual treatment—although I and my attendants could appreciate no improvement in hearing. Eventually she was convinced that she was making no headway.

If we conscientiously treat our deaf patients in a thorough manner, I feel that we would be much more satisfied with our results than by resorting as a routine to a questionable form of treatment such as the Zünd-Burguet electro-phonöide method.

## Reports of Cases.

### TWO CASES OF INTUSSUSCEPTION.

By P. L. HIPSLEY, M.D., Ch.M. (Sydney), F.C.S.A.,
*Honorary Surgeon, The Royal Alexandra Hospital for Children, Sydney.*

#### Case I.

K.O., a male, aged two and a half years, was admitted to the Royal Alexandra Hospital for Children on December 16, 1930. The illness had commenced suddenly with diarrhœa fourteen days before admission. Two days after the onset blood appeared in the stools and after another two days vomiting commenced. Vomiting was intermittent until the day before admission. The child was brought to the out-patient department on account of severe abdominal pain, spasmodic in character. At the time of admission a sausage-shaped tumour could be felt in the region of the transverse colon; a diagnosis of intussusception was made. A rectal injection of saline solution was given and some constipated fæces were seen in the return, but there was no blood. After the injection the mass disappeared and the child seemed quite well. The following morning the child seemed to be in pain again and the mass was again palpable in the region of the transverse colon. A second injection was given and a large fæcal result was obtained. Again the mass disappeared completely after the injection. I saw the child several hours later and he appeared quite well.

The following day the tumour reappeared and disappeared after another injection. On the two following days the child was quite well and the bowels operated several times normally. On December 20 the tumour appeared again and, by abdominal palpation, it could be felt to diminish in size until it finally disappeared. It was, however, deemed advisable to open the abdomen, and this was done by Dr. T. Y. Nelson.

*Operation.*

Within the abdomen there was evidence of a recent intussusception, in the thickening and congestion of the caecum and terminal portion of the ileum, but the intussusception had been completely reduced. Dr. Nelson described the presence of an abnormal fold of peritoneum at the ileo-caecal junction which he thought might have been responsible for the trouble. The appendix also was congested and was therefore removed. Since the operation there has been no further trouble.

*Comment.*

In this patient the intussusception had recurred several times during the six days whilst he was under observation in hospital; very probably it had been present off and on during the two weeks prior to admission.

Cases of this kind are seldom seen in children under two years of age. Still[1] mentions a similar occurrence in a boy, aged three and a half years; the intussusception was spontaneously reduced after symptoms had been present for three days, and in this case the intussusception recurred three months later and again disappeared after a few days' medical treatment.

#### Case II.

CASE II.—A.S., a male, aged nine months, was admitted to the Royal Alexandra Hospital for Children on April 17, 1930, with a history of intussusception of twenty-two hours' duration. The greater part of the intussusception was reduced by means of an injection of saline solution with the container at a height of 91 centimetres (three feet). The abdomen was then opened by a lateral muscle-splitting incision and the remainder of the intussusception was reduced. The complete reduction was only accomplished after a good deal of manipulation. The intussusception had commenced in the ileum, at a point about 25 centimetres (ten inches) from the ileo-caecal valve. After reduction the terminal portion of the ileum was dark in colour, but not actually gangrenous. The child was very ill for three or four days, while the temperature varied from 38·4° to 39·5° C. (101° to 103° F.), but he eventually settled down and left hospital at the end of two weeks, when he appeared to be quite well.

There were no further symptoms for the following three months. On July 16, 1930, soon after taking food, the child became suddenly attacked by pain and vomiting. The bowels did not operate for two days, but no blood was passed *per rectum*.

At the time of his admission to hospital the child was extremely ill and presented all the signs of general peritonitis. Dr. Keith Smith, who saw him in hospital, decided to operate. There was free pus in the abdomen and drainage tubes were inserted in both flanks and in the original wound. The child died several hours later.

*Post Mortem Examination.*

There was a fibrous stricture which almost completely occluded the ileum at a point about 25 centimetres (ten inches) from the ileo-caecal valve, that is, at the point where the original intussusception had commenced. Immediately above the stricture there was a diverticulum about the size of a walnut; in the diverticulum was a small gangrenous area, at the centre of which was a small perforation. The diverticulum was surrounded by adhesions which had fixed it to the transverse colon.

*Comment.*

The diverticulum was not present at the time of the original operation, hence it was due to the gradual yielding of the wall of the ileum above the stricture. The fibrous stricture was evidently due to contraction following on a ring of ulceration caused by the tightness of the intussusception.

Some years ago I had an opportunity of examining the interior of the ileum of a child who had died soon after the reduction of a very tight intussusception; in this case there was a ring of ulceration around the ileum a few inches above the ileo-caecal valve; no doubt it would have led to stricture if the child had lived. The case above recorded is, however, the only one I have seen in which almost complete occlusion of the bowel followed the reduction of an intussusception.

#### Reference.

[1] Goodhart and Still: "Diseases of Children," Ninth Edition, page 159.

### RUPTURE OF THE INTESTINE.

By JOYCE H. W. WHARTON, M.B., Ch.M. (Sydney),
*Monto, Queensland.*

P.G., a male, aged nine years, on June 22, 1930, when climbing up a railway cutting, pulled down a sleeper, which struck him on the head. As he fell down the cutting he struck his abdomen on a sharp projecting rock. It was reported to me that when found he was very shocked and complaining of intense abdominal pain and that he had had a hæmatemesis. He was brought to me twenty-four hours later suffering from intense shock. He was pale and frightened in appearance. His abdomen was distended and exquisitely tender all over. He protested at the lightest touch. There was a bruise in the left lumbar region at the site of maximum tenderness. His pulse rate was 140 and his respiration 24 per minute; his temperature was 36·7° C. (98° F.).

A diagnosis of subcutaneous rupture of the intestine was made.

About an hour later a laparotomy was performed under open ether anæsthesia. A longitudinal incision was made over the left rectus muscle which was retracted medially. When the peritoneal cavity was opened there escaped a considerable amount of clear fluid, also fragments of food and some silver paper off a chocolate. The patient then became pulseless, but revived after the administration of strychnine, the abdominal wound meanwhile having been covered with hot sponges.

Examination quickly revealed a mangled piece of small intestine about 7·5 centimetres (three inches) long. There was some slight attempt at adhesion formation. The torn ends were clamped and the macerated portion cut away. At this stage the patient again became pulseless and was again restored by means of strychnine.

After mopping up as much of the free fluid as time permitted an end-to-end anastomosis was quickly performed and the wound then closed in layers, a tube having been placed at either end of the wound, one to drain the loin, the other the pelvic cavity. Half a litre (one pint) of hot saline solution was administered *per rectum*.

During the next four days the patient's condition remained very bad. He was in much pain and was very restless, obtaining relief only from the administration of morphine. His pulse remained at a rate of 140 to 150 per minute and his temperature ranged from 36·1° to 36·7° C. (97° to 98° F.). The wound drained freely, at first, mostly a watery fluid. About the fourth day vomiting became troublesome, although it had until then been almost absent. Distension was still pronounced. It seemed inevitable that general peritonitis should supervene.

The following day he had a natural bowel movement, his temperature rose to 38° C. (100·4° F.) and gradually his general condition improved.

By about the eighth day his pulse rate was about 100 per minute and his evening temperature was round about 37·8° C. (100° F.). From then on his condition improved steadily, but there was copious purulent discharge from both tubes. By the end of the third week he had practically no pain except occasionally on micturition.

At the end of a month, as his temperature was not yet normal and his wound discharged only slightly, I feared that there might be a pocket of pus not draining. I therefore consulted with Dr. O'Regan who examined him for me under an anæsthetic. Digital exploration in the drainage tracks revealed nothing, but the dilatation caused them to drain freely for about two days. The discharge then ceased and the temperature fell to normal.

The patient was in all seven weeks in hospital, the remainder of the time being required for the healing of his wound. He went home in very good condition, with no complaints, and is seen at regular intervals. He is well and apparently suffers no ill effects from his experience.

This case seemed somewhat remarkable in the completeness of recovery considering the extensive trauma and the long interval which was allowed to elapse before medical attention was sought.

## INTRACRANIAL ARTERIO-VENOUS ANEURYSM.

By R. C. WINN, M.B., Ch.M. (Sydney),
*Honorary Assistant Physician, Sydney Hospital, Sydney.*

THE patient, Mrs. O.G., aged sixty years, recovered from all her symptoms and signs in June, 1929. This fact tends to confirm the diagnosis of intracranial arterio-venous aneurysm, which was suggested in 1928, when she was shown at the Medical Science Club and the Sydney Hospital Clinical Society. Her symptoms then were: (i) A continuous roaring noise in the head, loudest on the left side, which had existed for three years; (ii) attacks of giddiness, the first and worst four years previously; (iii) a "tight feeling" at the left occipital region of the skull for three months; (iv) loss of sleep and defective memory for three years; (v) emotionalism, phobias and attacks of visual hallucinations in a kaleidoscopic series (phantasmagorias), worse for the previous years but occurring at intervals since childhood.

When the stethoscope was applied anywhere on the patient's head an intensely loud roaring noise could be heard, loudest over the region of the left mastoid process. This noise was continuous, but was accentuated during cardiac systole. Pulsations of the left occipital artery were much more easily palpable than those of the right. The mastoid branch of the occipital artery which enters the mastoid foramen and is described in Cunningham's "Anatomy" as "a small and inconstant branch," appeared to be nearly as big as the normal occipital artery. The noise could be almost completely obliterated (as judged by the patient or by listening with a stethoscope) when firm pressure was made over the left common carotid artery. The noise was greatly reduced by firm pressure over the left occipital artery or its mastoid branch. If light pressure were exerted over the occipital artery a whistling sound would arise.

The systolic blood pressure was 120 and the diastolic 80 millimetres of mercury.

No abnormality was found by means of X ray examination of the skull, the Wassermann test, a full blood count, examination of the ocular fundi and of the eighth nerve.

### Comment.

A diagnosis of arterio-venous aneurysm was made because of the nature of the noise and its intensity. The bruit of an arterio-venous aneurysm is continuous with systolic accentuation. The absence of obvious venous dilatation is not against such a diagnosis, because there is usually no obvious venous dilatation when the arterio-venous aneurysm is in the head region.

Actually the hypertrophy of the left occipital artery may have been more apparent than real and its pulsation may have seemed to be in excess of the normal. This might conceivably occur if the *venæ comites* of the occipital artery were dilated by back pressure.

That this arterio-venous aneurysm was intracranial was probable because of the site of the bruit. Most intracranial arterio-venous aneurysms occur between the internal carotid artery and the cavernous sinus. As there was no proptosis (or even choked disc), as the noise was loudest over the left mastoid process and as the "tight feeling" was over the left occipital region, it was probable that the aneurysm was not at the usual site, but in the posterior cranial fossa on the left side.

The phantasmagoria might have been due to stimulation of the occipital region of the cortex. Though the attacks had increased in frequency since the noise commenced, they had existed since childhood. Such a change might be accounted for by postulating a congenital intracranial aneurysm (such as is stated to have been found at one in every 125 of over 5,000 consecutive *post mortem* examinations of the brain at the London Hospital) which had ruptured into a vein three years previously.

Congenital intracranial aneurysm is more likely to produce a bruit if it affects the basilar and vertebral arteries than if it is in a more anterior position.

The attacks of giddiness were probably due to changes in the blood supply (perhaps to the semicircular canals as well as to the brain). It was during an attack of giddiness that the symptoms and signs disappeared. Probably complete obliteration of the aneurysm occurred at that instant on account of a sudden obstruction, by a tag of clot, to the outflow from the cavity.

It is difficult to explain the subsequent disappearance of the apparent hypertrophy of the left occipital artery, unless it be assumed that the difference between it and the right occipital artery was due to dilatation of its *venæ comites*; obliteration of the aneurysm would lead not only to disappearance of this dilatation but also to collapse of these veins.

This patient was shown at the clinical meeting of the Medical Science Club held on June 2, 1930.

## CARDIAC RUPTURE.

By D. F. O'BRIEN, M.B., F.R.C.S.I.,
*Rockhampton.*

UNDER the caption "Current Comment," an article headed "Cardiac Rupture" appeared in THE MEDICAL JOURNAL OF AUSTRALIA of February 7, 1931. The following notes on a case of cardiac rupture will be of interest.

J.W., an old age pensioner, aged eighty-two years, while having a sharp argument with a neighbour, dropped at the latter's gate and died at once, on April 9, 1930.

### Post Mortem Examination.

The body was pale and spare and bore no marks of violence. The pericardium was full and slightly distended with serum and blood; it contained no clots.

There was a round hole in the anterior wall of the left ventricle, half way from base to apex, and 1·25 centimetres (half an inch) from the septum. I had recently examined a bullet wound of the heart and this at first sight appeared similar. But closer examination revealed that the edges of the hole were broken in and sharply defined and that the hole was not perfectly round. It was not punched out like a bullet wound.

A little nearer to the base and a little further from the septum there were cracks in the visceral pericardium, of a similar shape and size, but with the underlying muscle still *in situ* and evidently partly torn inwards. The left ventricle was empty. The plug of muscle corresponding to the first mentioned opening was attached to an anterior papillary muscle. The second (partial) rupture was at the site of attachment of another papillary muscle.

The orifice of the anterior coronary artery was atheromatous. There was no thinning of the cardiac wall, but it was friable. The size of the heart was normal.

A piece of parchment-like paper was found fixed in the duodenum. It had caused an ulcer opposite the entrance of the bile duct into the duodenum. Nothing special was observed in other organs.

# Reviews.

## EXAMINATION OF THE NERVOUS SYSTEM.

"NEUROLOGICAL EXAMINATION," by Dr. C. A. McKendree, fills a much needed want in medical literature as an introduction to neurology.[1] The practitioner and senior student will find particular use for this work as a guide to the systematic investigation and examination of neurological cases. Signs and symptoms are interpreted in terms of modern anatomical and physiological principles.

The first chapter deals with the history taking and general methods of approaching a neurological case to be investigated. A very detailed and comprehensive case sheet is illustrated.

Abnormalities of the motor functions of the central nervous system are discussed in the next chapter. The attitude, gait, muscle coordinating and performance of skilled acts are dealt with, and pathological conditions affecting these states of body are lucidly explained.

Abnormal involuntary movements, such as tremors, twitching, athetosis and choreiform movements, are explained on physiological bases. Such explanations are necessarily abbreviated, and it is obvious that their significance cannot be fully appreciated without a sound grounding in the anatomy and physiology of the central nervous system.

Reflex activities are explained by means of diagrams, and the various methods of eliciting reflexes are described. The clinical significance of abnormal reflexes is fully discussed.

Loss of muscle power, abnormal muscle tonus and muscle wasting and their significance are discussed. The physiological principles, laid down by Sherrington and his collaborators, are unfortunately not made use of in the explanation of reflex activities and muscle tonus.

A separate chapter is devoted to the sensory approach to neurological examination. Instruments for measuring qualitative and quantitative sensory disturbances are illustrated. The anatomical distribution of sensory tracts and the clinical manifestation of their loss of function are briefly detailed.

The chapter on the examination of the cranial nerves is well illustrated and clinical syndromes associated with various cranial nerve palsies are aptly described. A short description is given of some of the more common general medical clinical conditions found in association with neurological conditions. A brief indication of the methods of approaching psychiatric patients is described, and, lastly, an outline of laboratory tests used in neurological diagnosis is given.

The book consists of 250 pages profusely illustrated, both by diagrams and photographs.

[1] "Neurological Examination," by Charles A. McKendree, A.B., M.D., with a Foreword by H. A. Riley, A.M., M.D.; 1928. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Post 8vo., pp. 280, with illustrations. Price: 16s. 6d. net.

## SURGERY OF THE EAR.

"OTOLOGIC SURGERY" is now in its second edition and has been brought up to date; the chapter on laboratory aids has been extended.[1] The author attempts a big task when he claims the book to be suitable for the student, medical practitioner and specialist. It lacks in illustrations and is not sufficiently understandable by the two former.

From the point of view of otological pathology the work is well worth possessing, being both instructive and interesting; the many references to authorities on the various subjects show much thought and discrimination. The book should find a useful place in every otologist's library.

## DIATHERMY IN OTO-LARYNGOLOGICAL PRACTICE.

IN view of the widespread use of diathermy and its special application to diseases of the upper respiratory tract Dan McKenzie's book on "Diathermy in Otolaryngology" does not come as a surprise.[2] As is usual in McKenzie's writings the material in this book is drawn largely from the author's own experience; but in those cases in which he feels that he cannot speak authoritatively, he pays graceful tribute to the work of others.

The book is well printed, typographical and grammatical errors are very few in number and the illustrations, though neither very numerous nor elaborate, are clear. The subject matter is dealt with in detail and with great clarity, so that the book makes easy reading. One of the most valuable chapters is that on ineradicable cancer. All those who have had any experience in the treatment of this type of cancer by diathermy, will appreciate the author's view that in diathermy we have a palliative agent of the very greatest service.

For an enthusiast McKenzie is singularly unbiased in his claims, and he has no hesitation in expressing an adverse opinion on diathermy when he feels that the occasion demands it. His few words on the relative value of radium and diathermy contain the following passages, which sum up the situation: "At the present moment we are unable to say which is the more life saving remedy," "we can employ radium in combination with diathermy with great benefit," "thus the wise plan is to make use of both remedies."

This is an excellent little book which, from the "value for money" point of view, is a pleasant change from the more highly priced and elaborately produced monographs to which we have become accustomed.

[1] "Otologic Surgery," by Samuel J. Kopetzky, M.D., F.A.C.S.; Second Edition, Revised; 1929. New York: Paul B. Hoeber, Incorporated. Royal 8vo., pp. 570, with illustrations. Price: $8.00 net.

[2] "Diathermy, Medical and Surgical, in Oto-Laryngology," by Dan McKenzie, M.D., F.R.C.S.E.; 1930. London: Kegan Paul, Trench, Trubner and Company Limited; Melbourne: W. Ramsay. Demy 8vo, pp. 198, with illustrations. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, MARCH 28, 1931.

*All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.*

*References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

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## AN ASSOCIATION OF PHYSICIANS.

In a recent issue of this journal there appeared a short account of the inauguration of The Association of Physicians of Australasia (including New Zealand). As stated in the published report, the founders of the Association wished to model the new body on The Association of Physicians of Great Britain and Ireland, "which is purely scientific in its scope and engages in no medico-political activities." The objects of the Association are "the advancement of internal medicine and the promotion of friendship among physicians." The qualifications for membership are set out in the statement that the Association "shall consist of Ordinary Members, not more than ten Associate Members resident in New Zealand, and Honorary Members. It shall at first contain not more than seventy Ordinary Members who shall be physicians actively engaged in the teaching of medicine, men of distinction in medicine and the allied sciences, and physicians recognized by the Council as eligible." Meetings of the Association will be held in capital cities twice a year and practically the whole of the time available at the meetings will be devoted to scientific communications by members and visitors. Communications are to be spoken and not read. No representatives of the press, lay or medical, will be present, and no report of the proceedings will be sent to medical journals or newspapers. Members will be allowed to publish their communications in whatever journals they choose.

The new association will be welcomed. Since the foundation of the College of Surgeons of Australasia which includes New Zealand (to give the College its full and official title), many persons have hoped that the physicians of Australia and New Zealand would band themselves together in some organization of a similar nature. The suggestion has been made that the inauguration of The Association of Physicians of Australasia is the first step towards the foundation of a College of Physicians. There is no indication that this is so, and no suggestion of such a kind has been made by the founders. At the same time, if a College of Physicians of Australasia is to arise, and we are sincere in hoping that such a college will eventually be formed, it will probably come into being by evolution of the Association of Physicians of Australasia. It is a little unfortunate that the new association has seen fit to perpetuate the pleonasm introduced by the College of Surgeons of Australasia by attaching to the title the words "including New Zealand." Everyone who knows anything at all about geography knows that Australasia includes New Zealand. It would be just as sensible to talk about the medical practitioners of New South Wales including Sydney, or those of Melbourne including Collins Street. This, however, is but a small point. It is not the name, but actions that will count. For the objects of the association, the arrangements for meetings and the qualifications for membership we have nothing but praise. The limitation of membership to a small number of persons is wise. There is a danger that new associations may make the opening of their doors too wide, that by increase in numbers a high standard may be lowered and that membership will not be highly regarded. Of course personal animus must never be introduced—attention must be focused only on scientific attainment. The names of the office bearers, under the presidency of Sir Richard Stawell, are sufficient guarantee that election to membership will be made with wise discrimination. Scientific attainment must be considered apart from seniority, for the latter is one of the greatest bars to progress when it is used as

an argument against the advancement of preeminent juniors. The regulation prohibiting the routine reading of papers is excellent. A similar rule might be adopted with advantage at some Branch meetings. Discussion is more likely to be useful when an author merely speaks to his subject, and much of the long-winded description of the work of other authors will be eliminated. The most useful feature of the activities of the new body will be the more frequent exchange of views between leading physicians of the several States. This has been found, from the point of view of the individual, to be the most useful feature of the work of the College of Surgeons of Australasia.

The main object of the new association is the advancement of internal medicine. This will, of course, depend on the individual and in these matters the individual cannot be considered apart from his ethical standards. Both physicians and surgeons in Australia who set themselves up as specialists, would do well to pay some attention to the limitation of their private practice to the specialties which they profess. The spectacle of senior physicians and senior surgeons on the staffs of large hospitals (to say nothing of juniors) engaging respectively in surgical and medical practice outside the hospital, is not edifying and does not inspire confidence. This should be seriously considered by both the College of Surgeons of Australasia and the Association of Physicians of Australasia.

## Current Comment.

### "UROSELECTAN."

PROGRESS in urology has been hampered to some extent for want of a simple method of permitting radiographic definition of the urinary tract. True, pyelography by means of the local injection of a radio-opaque substance has been commonly practised, but its performance requires special knowledge and skill and it can scarcely be termed a simple procedure. Thoughtful workers have been seeking a non-toxic radio-opaque substance which, after its administration—perhaps orally, perhaps intravenously—is eliminated by the kidneys in a sufficiently strong concentration to throw a shadow on the X ray film. In 1923 Rowntree and his collaborators showed that pyelography was possible after the intravenous injection of a solution of sodium iodide; their experiments were not completely and invariably successful, however, and it was evident that further research was necessary and that the ideal substance for injection was yet to be found. The next advance was made when Roseno clinically administered a substance formed by the combination of urea and iodine and successfully examined the urinary tracts by means of X rays. The substance used by Roseno, however, was not tolerated by all patients; furthermore, its excretion was so slow that pyelography after its administration was not possible in every instance. The work of Binz and Räth in chemistry and of Hryntschak, Lichtwitz, von Lichtenberg and Swick in the clinical application of pyelography led eventually to the evolution of "Uroselectan," the sodium salt of dioxy-pentaiodo-pyridine-nitro-acetic acid. It contains 42% by weight of iodine and is said to be non-toxic. It is injected intravenously in a 40% solution and is eliminated in the urine, often, it is said, in a concentration of five *per centum*.

Recently "Uroselectan" has been discussed in three separate papers by Alexander von Lichtenberg, by Arthur Binz, and by R. A. Gardner and R. St. A. Heathcote.[1] The history of "Uroselectan" and its clinical application is outlined by von Lichtenberg. He is convinced of its practical importance and its safety. He remarks that, apart from the value of "Uroselectan" as a contrast substance, the manner of its excretion may provide knowledge concerning the condition of the kidneys, for the rapidity of its elimination depends upon good renal function. He admits, however, that little information in this regard may be obtained, save when the kidneys are severely damaged. It would appear, therefore, that nothing can be learned concerning the kidney function beyond what may be readily ascertained by the employment of less elaborate procedures. According to von Lichtenberg, a dose of one hundred cubic centimetres of a 40% solution of "Uroselectan" is suitable for purposes of pyelography.

Gardner and Heathcote, of the Departments of Radiology and Pharmacology at the Egyptian University, determined to undertake experimentation with animals before administering to human subjects "Uroselectan" in such a large dose as forty grammes, or 0·5 to 0·66 gramme per kilogram of body weight. Assuming that the blood volume of the average man be 5,000 cubic centimetres and that the whole dose of "Uroselectan" were in circulation at the same time, there would be a 0·8% solution of the drug in the blood.

By means of Gunn's apparatus, Gardner and Heathcote perfused the isolated heart of a rabbit with a 0·8% solution of "Uroselectan" in Locke's solution. They report that the beat of the heart was depressed by this means, but there was no appreciable alteration in the rate nor in the outflow from the coronary orifices. Effects were more pronounced when a stronger solution was employed, but a true evaluation could not be made on account of the changes brought about by increased osmotic

[1] *The British Journal of Urology*, December, 1930.

pressure. It was found that the action of "Uroselectan" on the isolated intestine of the rabbit resulted in a depression of the muscular contractions of the organ. Gardner and Heathcote gathered that the effects of the solution on both heart and intestine were due to its direct influence on the involuntary muscle fibres.

Next, a solution of "Uroselectan" was administered intravenously to dogs, and the effect on the blood pressure observed. In the first experiment a dose of 3·3 grammes per kilogram of body weight (five times the proportionate human dose) was administered without regard to the rate of injection. The result was a rapid fall of blood pressure from 105 to 30 millimetres of mercury, followed by the death of the animal. There were great congestion of the organs and acute œdema of the lungs. In subsequent experiments the drug was injected more slowly, though still in large doses. On each occasion its administration was followed by a considerable fall in blood pressure. Nevertheless, it was concluded that large doses were well tolerated by rabbits and dogs, providing the injections were slowly made. Several intact and normal rabbits were killed by too rapid injection. Dogs rendered nephritic by the injection of uranium nitrate did not excrete any of the drug by way of the kidneys.

The result of one experiment tended to show that the elimination of "Uroselectan" from the general circulation is very rapid, as, sixty minutes after the injection, only a trace of iodine could be found in a sample of blood withdrawn. There is, however, a probability of fallacy here, as undoubtedly "Uroselectan" is still being excreted by the kidneys some hours after the injection.

When "Uroselectan" was administered to dogs in the human dosage (0·66 gramme per kilogram of body weight) the kidney outlines observed by means of X rays were no clearer than before the administration of the drug, but when a dose of 4·0 grammes per kilogram was given, excellent skiagrams of the renal calices, ureters and bladder were obtained. The best skiagrams were made four hours after the injection; there were no shadows nine hours after the injection. This is interesting in view of von Lichtenberg's recommendations that skiagrams should be made one-quarter of an hour, three-quarters of an hour and one hour and a quarter after the injection. Possibly elimination is more rapid in man than in the dog.

Gardner and Heathcote conclude that "Uroselectan," administered slowly in a dosage as high as 6·0 grammes per kilogram of body weight, is innocuous to rabbits and dogs and that it renders the urine of dogs opaque to X rays, with the result that satisfactory skiagraphy of the urinary tract is possible.

It is apparent that much remains to be learned concerning the pharmacology of "Uroselectan." While the presence temporarily in the circulation of 16·8 grammes of iodine may perhaps be regarded with complacency, there may be an excuse for scepticism concerning the alleged harmlessness of its more or less prolonged retention when the kidneys are unable to excrete it. Gardner and Heathcote make no mention of the paths of elimination in these circumstances. However, there can be no doubt that, judiciously employed, "Uroselectan" is a valuable aid to diagnosis.

Pyelography subsequent to the injection of "Uroselectan" has certain obvious advantages over the instrumental instillation of a radio-opaque substance into the ureter; furthermore, it is applicable in certain conditions in which the other method is not. But the drug can be administered directly in far stronger solution than it can be excreted by the kidneys; the application of stronger solutions, of course, results in clearer radiographic definition. The relative advantages and disadvantages of the two methods should be weighed in each instance, neither one nor the other should be employed as the result of a metaphorical "spin of the coin." The advent of "Uroselectan" opens up a new field of research for the radiologist. It may be safely assumed that he will not be tardy in his work of exploration.

It is unfortunate that the advent of important discoveries in medical science should be so frequently accompanied by a terminological atrocity; in this instance it is "intravenous urography." No doubt the value of the advancement outweighs the tragedy of the slaughter of the mother tongue, but the slaughter is unnecessary and unkind.

## CURE IN PERNICIOUS ANÆMIA.

An important report has recently been published by A. F. Hurst of a case in which the gastric secretion of a patient with pernicious anæmia became normal after treatment.[1] The patient, a man of seventy, gave a history which suggested that his achlorhydria might be secondary to chronic gastritis. He also suffered from *pyorrhœa alveolaris*. The patient's clinical picture was typical of pernicious anæmia. The patient was given liver extract, his stomach was washed out with hydrogen peroxide and his pyorrhœa was thoroughly treated. Hurst states that only one similar case, as far as he knows, has been recorded. Achlorhydria, which is an essential predisposing cause of pernicious anæmia, is not necessarily due to constitutional *achylia gastrica*. It may result from achylia secondary to gastrectomy or atrophic gastritis and also from achlorhydria due to chronic gastritis in which atrophy of the mucous membrane is not necessarily present. It may be concluded that Hurst's patient was cured; he will not have to continue the use of liver extract. The lesson to be learned is that when the history indicates that the achlorhydria may have resulted from gastritis, when excess of mucus is revealed by the test meal and when there is no family history of pernicious anæmia, an attempt should be made to restore the secretion of a normal gastric juice. Only in this way is cure possible.

[1] *Guy's Hospital Reports*, October, 1930.

# Abstracts from Current Medical Literature.

## BACTERIOLOGY AND IMMUNOLOGY.

### The Preparation of Antigens from Cultures of Treponema Pallidum.

AUGUSTUS B. WADSWORTH, JEANNE E. VAN AMSTEL AND MARGARET W. BRIGHAM (*Journal of Immunology*, September, 1930) detail experiments in the preparation of antigens from *Treponema pallidum* with a view to studying the nature of the complement fixation reaction in syphilis. The strain of *Treponema pallidum* used was originally obtained from Noguchi and kept alive in Smith tissue medium through successive transfers since 1924. After experimentation with several kinds of media the following was adopted. Culture flasks half full of a mixture of equal parts of normal horse serum and beef-infusion broth (with pieces of autoclaved guinea-pig liver at the bottom of the flasks) were placed in an Arnold sterilizer at 100° C. for forty-five minutes, in which time this medium becomes firmly coagulated. The flasks were then filled with sterile uncoagulated medium of the same composition with the exception of the liver. From the liquid portion of this medium the treponemata were readily separated by centrifugalization. The great practical difficulty of cultivating treponemata in any but very small amounts was not overcome. The results obtained by the authors agree essentially with the observations of Noguchi. By the aqueous extraction of the organisms antigenic substances were obtained which react in a marked degree with the serum of rabbits immunized with the same cultural strains, but not with syphilitic rabbit or syphilitic human sera. The addition of cholesterol to the aqueous, strong salt and alcoholic extracts of *Treponema pallidum* did not increase their sensitivity, but in the case of the alcoholic extracts especially it rendered them extremely anticomplementary. The results obtained with dialysed aqueous extracts of *Treponema pallidum* which had been previously extracted with alcohol, indicate that the antigenic substances obtained by aqueous extraction of the organism differ from those obtained by extraction with alcohol. Antigenic substances which reacted with rabbit immune serum, syphilitic rabbit and syphilitic human sera were obtained by the extractions of the uninoculated medium with alcohol. Slightly increased antigenic activity was noted with similar extracts of the whole culture medium containing *Treponema pallidum*. These observations suggest that the divergent results reported in literature concerning the solubility in alcohol of antigenic substances of this organism may be due to differences in the media used and the culture materials which were extracted. *Treponema pallidum* produced, in culture, substances which fixed complement in the presence of very small amounts of the sera of rabbits immunized with the homologous strain, and the activity of these substances corresponded to that obtained by aqueous extracts of the treponema free from medium.

### Poliomyelitis.

R. W. FAIRBROTHER AND W. G. SCOTT BROWN (*The Lancet*, October 25, 1930) record results of investigations which support Aycock's view that the observed immunity of adults to poliomyelitis is most probably due to "subclinical" infections. The authors' attention was directed to the fact that serum of a normal individual might possess antiviral activity when, in order to supply an extra control of the antiviral activity of several samples of convalescent serum, a sample of normal serum was also mixed with the test virus. The animal inoculated with the saline control became infected and died while all the others remained well. Later a small outbreak of bulbar poliomyelitis which occurred in a private boarding school, provided an opportunity of continuing the study. Some time after the attack had occurred in the school, samples of serum were obtained from convalescent patients and those with abortive infections and from close and remote contacts. The findings were similar to those obtained by the American workers. The control animal became infected after an incubation period of five days, while all the other animals remained quite well, indicating that the serum in all cases contained antiviral bodies. A degree of immunity was found to be present in contacts who had never had the slightest sign of poliomyelitis, but whether this immunity was the result of contact with definite infections in this outbreak or had been acquired at some previous period could not be decided. The sera of three persons who had never, so far as could be ascertained, been in contact with a case of poliomyelitis, were also examined. One of these sera was proved to contain antiviral bodies, the other two did not.

### Persistent Eosinophilia.

A. M. DRENNAN AND J. H. BIGGART (*Journal of Pathology and Bacteriology*, October, 1930) give details of the study of a case of persistent eosinophilia with splenomegaly. The patient was a school boy, aged fifteen years, had never been abroad and gave no history of helminthic infection. At the first examination the blood count revealed 2,360,000 red cells per cubic millimetre with 50% hæmoglobin value, 61,250 leucocytes per cubic millimetre and 29% eosinophile cells. Four days before death the red cells numbered 1,100,000 per cubic millimetre, the hæmoglobin value was 20%, leucocytes numbered 70,000 per cubic millimetre with 70% eosinophile cells. Injections of splenic extract were administered in an attempt to reduce the eosinophile cells. After three injections of five cubic centimetres of 40% splenic extract the total number of eosinophile cells fell from 23,853 to 7,902 per cubic millimetre, and after the injections were stopped they rose to 18,200 per cubic millimetre. During the period of injection the patient appeared to improve slightly, but their cessation was soon followed by death. *Post mortem* the spleen was slightly enlarged with several projecting nodules on the surface. A pelvic tumour on the inner and lower aspects of both psoas muscles involving the iliac vessel and ureters was noted. Lymph glands, examined histologically, were found to be diffusely infiltrated with eosinophile cells. The tumour in the pelvis consisted of striped muscle with gross cellular infiltration consisting preeminently of mature eosinophile cells with lymphocytes and numerous macrophages. The bone marrow showed the majority of cells to be eosinophile cells, both mature and myelocytic and intermediate. Polymorphonuclear cells were in diminished numbers and there was very little red cell formation. Except in the bone marrow all the eosinophile cells were of mature type. The majority had bilobed nuclei, but cells with three, four, five or even six lobes were not uncommon. The authors consider that the condition is one of excessive response of normal eosinophile cells to some unidentified irritant rather than a primary neoplastic or leuchæmic infiltration. The majority of the eosinophile cells in this case, as well as in the eleven other reported cases tabulated by the authors, raises the question of the justification of classifying the condition with the leuchæmias.

### Bacillus Typhosus in Vaccines.

FRANCIS B. GRINNELL (*Journal of Immunology*, November, 1930) carried out a series of experiments to test the relative value of vaccination with rough and smooth strains of *Bacillus typhosus*. The bactericidal power of the blood of individuals who had been immunized with the smooth and rough vaccines, was compared. It was found that the bactericidal power of the blood is very little, if any, increased by the customary course of treatment with a rough vaccine, while a similar treatment with a smooth vaccine has a pronounced effect. The fact that the rough vaccine produces an increase in agglutinins for the virulent strain comparable with that produced by the smooth vaccine, and the fact that the bactericidal strength fails to parallel the agglutinin titre would indicate that agglutination is not an adequate test of the degree of immunity of the individual.

### Resistance of Guinea-Pigs Vaccinated with Bacille Calmette-Guérin.

S. A. PETROFF AND WILLIAM STEENKEN, Junior (*Journal of Immunology*, July, 1930) compare the resistance established in guinea-pigs vaccinated intraperitoneally, subcutaneously and perorally with living *Bacille Calmette-Guérin*, heat-killed H37, and living Ri organisms. It was found that the

resistance established by *Bacille Calmette-Guérin* vaccination was in no way greater than that established in animals vaccinated with heat-killed tubercle bacilli. The animals vaccinated with living Ri apparently developed a slightly greater protection than either of the others. Animals vaccinated intraperitoneally or subcutaneously with living *Bacille Calmette-Guérin* developed some degree of protection. Guinea-pigs vaccinated by feeding exhibited no evidence of allergy after the lapse of a period of four weeks, and no perceptible immunity. The results obtained confirm Langer's observations that the resistance established with *Bacille Calmette-Guérin* is not greater than that established with heat-killed bacilli.

## HYGIENE.

### The Heating of Buildings.

H. M. VERNON (*Journal of State Medicine*, September, 1930) describes the new method of heating buildings by low temperature radiation. The principle consists in warming large surfaces or panels placed in the ceilings or walls of the room to about 37·8° C. (100° F.) by concealed hot water pipes or electric resistance wires. Heat from these panels radiates and warms the surfaces against which it strikes. The heat imparted to the air in contact with these surfaces is of so mild a character that the surfaces are only about 3° C. warmer than the surrounding air. Steel pipes of 1·25 centimetres (half an inch) internal diameter are used, placed in rows or coils about ten centimetres (four inches) apart. These are embedded in the ceiling or wall plaster and are invisible. Usually a band of pipes 90 or 120 centimetres (three or four feet) in width runs around the periphery of the ceiling, which is a better position than the walls. Forty to fifty *per centum* of heat transmission from walls is by convection from the rising of heated air currents; from the ceiling the heating is almost entirely by radiation. Repair of concealed water pipes is difficult. Accordingly, a system of electric heating by panels has been used. These contain insulated wires embedded in plaster on a cork slab and secured to cement asbestos backboards. Temperature is controlled by a thermostat. A still more recent method has been employed by using fire-clay plates with a zig-zag ribbon of graphite running through. By sending a current through the graphite the plate is warmed to 260° C. (500° F.). To distribute radiation it is advisable to keep these as high as possible in the room, thereby preventing distribution of heat by convection. Experience has shown that panels of somewhat over 37·8° C. (100° F.) give the optimum radiation. The amount of heat from convection which is desirable will depend on various factors, as the type of occupation *et cetera*. Convection heating is less fresh and exhilarating than heating by radiation. Under-floor heating by means of hot air ducts has been known since the Roman era in Britain. Recently schoolrooms have been equipped in Derbyshire with a system of under-floor hot water pipes. Ample ventilation by hopper windows and swing-back doors is provided. The warm floor is found to be pleasant if the temperature does not rise above 21·1° C. (70° F.). The maximum of comfort and efficiency in light sedentary work is estimated at between 15·5° and 18·3° C. (60° and 65° F.) with a dry Kata thermometer reading of six to seven. Testing the efficiency of school children showed the best results in the hot water radiator heated rooms. Where the temperature of class rooms falls below 15·5° C. (60° F.) there is a falling off in attendances, probably due more to disinclination than sickness. It is generally considered that the true open air school with little or no artificial heating has an extremely beneficial effect on the health of subnormal children, but the advantages would be enhanced by introducing under-floor heating in the winter. Probably the best method of heating school or work rooms would be by installing a combined under-floor and overhead panel system.

### Constitution in Health and Illness.

C. NAEGELI (*Journal of State Medicine*, May, 1930), commenting on the reduced incidence of infectious diseases in Zurich and elsewhere of recent years, points out a group of diseases in which endogenous predisposition by heredity is the most important factor, the constitutional diseases. Hæmophilia, certain nervous diseases, diabetes, arterial and cardiac diseases, all depend largely on this factor. However, the factor of heredity is a recessive, not a dominant factor, and public health measures may be directed to the control of these diseases. Certification of health prior to marriage would be of value, but unfortunately in this matter sentiment will prevail. At the same time, external influences very often are required to cooperate with the disposition to produce the disease, and these can be controlled by hygienic measures.

### Arterial Hypertension in Industry.

JAMES N. WYCHGEL (*Journal of Industrial Hygiene*, November, 1930), in conducting a physical examination of 354 employees fifty-five years of age and over, in one of the American steel plants, paid particular attention to the blood pressure. Of the total 73% were found to have hypertension according to the standard found by adding the subject's age to 100 for the systolic pressure. The normal limit of diastolic pressure was taken as 100. For the purposes of classification three degrees of hypertension were arbitrarily chosen. The first comprised men in whom the systolic pressure ranged between 150 and 170 millimetres of mercury or the diastolic pressure between 100 and 110 millimetres, or both, and 47% of the examinees were found to be in this group. Such men are advised to seek medical advice every six months and, if they do not improve under treatment in one year, should be regarded as belonging to the second degree. In the second group the systolic pressure recorded lies between 170 and 200 millimetres, or the diastolic from 110 to 120, or both; 22% were in this group. Cardiac hypertrophy is present. Activity should be limited to light work and the individuals should be examined every six months for cardiac failure. In the third group the systolic pressure is over 200 or the diastolic over 120 millimetres of mercury, or both; 4% of examinees were found in this group. Such persons are unfit for work. Classification of the results in quinquennial age groups showed no progressive increase of pressure with advancing years.

### Electric Shock.

WILLIS MACLACHLAN (*Journal of Industrial Hygiene*, October, 1930) has collected information in connexion with approximately 700 cases of electric shock in Canada and the United States over a period of twelve years. Certain cases were rejected owing to death occurring from causes such as broken neck, extensive burns *et cetera*, and the remaining 479 cases are divided as follows: The person receiving the shock becomes unconscious and stops breathing, resuscitation causes resumption of breathing and the patient lives—323 cases; under similar circumstances resuscitation fails and the patient dies—156 cases. The percentage of successfully treated persons is found to be 67 and the monthly distribution is found to vary in a curve which descends from March to September and rises again to December. The highest percentage of successful treatment thus occurs in the winter months. The influence of fatigue on success of treatment by resuscitation is shown by plotting the percentage of successes according to the time of day. There is a falling off in the afternoon curve. Comparing the results of treatment with the potential of current, a definite rise in percentage of successes with rise in potential is shown. Probably this is due to poor and brief contact in higher potentials. Of 282 victims who fell clear after contact, 70% were successfully treated. In 197 cases it was necessary to pull the victim clear, indicating prolonged contact; among these the percentage successfully treated was only 63. Promptness in application of resuscitatory measures is an important prognostic factor. This is shown by dividing the patients treated into two groups: one, those receiving treatment within four minutes of the shock, and the second, those receiving treatment four minutes or more after the shock. In the first group of 361, 70% recovered, while only 58% of the second group recovered.

# Special Articles on Diagnosis.

*(Contributed by Request.)*

## XXXVIII.
## ACUTE POLIOMYELITIS.

THE differential diagnosis of acute poliomyelitis covers a wide field of clinical medicine and, as the disease may affect adults, it should not be regarded as of interest to pædiatricians alone. In rural districts and among native races, many cases occur among young adults. It is unfortunate that the pre-paralytic stage of poliomyelitis is not heralded by any dramatic symptom, comparable to the warning symptoms, croup and stridor, of laryngeal diphtheria or to the severe spasmodic pain followed by pallor which terrifies the parent of a child suffering from intussusception. Poliomyelitis comes more quietly and the diagnosis at the stage at which specific treatment is effective is rendered still more difficult by the fact that this disease may occur at any age, and the symptom complex may be imitated by many other acute diseases. It is agreed, however, by everyone who has had the opportunity of seeing an epidemic of poliomyelitis, that clinically a definite picture is presented which makes a diagnosis in the pre-paralytic stage a possibility, if time be given to a careful examination and if certain signs are deliberately elicited. In no other disease we are called upon to treat, does so much depend upon the vigilance and acumen of the doctor, for while with other emergencies we gamble with death, with poliomyelitis the stake may be life-long crippling.

If we think of the disease as a general systemic infection, accompanied or followed by a myelo-encephalitis with meningeal complications, and if we appreciate the fact that the disease may affect any or all parts of the central nervous system in all possible combinations, we shall understand its variable manifestations and be less likely to overlook it or mistake it for some other condition.

The lengthening of the pre-paralytic stage of the disease, a fact which has been noted in the last two decades, has made the early recognition easier. We have not, in Australia, recently seen fulminating cases in which the onset of paralysis almost synchronizes with the initial sickness. The mode of onset has conformed rather to the dromedary or straggling types described by Draper. The greatest difficulty in diagnosis is the "time" factor. If a patient be regarded as possibly suffering from pre-paralytic poliomyelitis one is not justified in temporizing; a diagnosis must be made forthwith.

The "suspect" cases fall into two groups, namely:

I. The group in which the clinical examination renders examination of the cerebro-spinal fluid unnecessary.

II. The group in which examination of the cerebro-spinal fluid is required for purposes of differentiation.

The first group may be divided into divisions (*a*) and (*b*): (*a*) The cases in which poliomyelitis is suspected because of the patient's inability to use a limb, or because of the development of a limp; (*b*) the acute febrile diseases which may be imitated by the early stages of poliomyelitis.

### Poliomyelitis Suspected on Account of Loss of Efficiency of a Limb.

#### *Injury.*

A green stick fracture of the clavicle, radius or fibula of a child, a sprain of the ankle or knee, may cause no local swelling or ecchymoses, but be manifested by an apparent loss of power or disinclination for normal activity. The absence of temperature, of any history of antecedent illness, the absence of any other evidence of poliomyelitis, such as retention of urine, tremor, the spine or Amoss's sign, combined with definite localized tenderness to palpation, will usually differentiate and an X ray examination may confirm. When the complaint is inability to stand alone or to use a limb, or a limp of recent development, observation of the erect posture adopted by the child helps one to decide whether the limb is painful only or painful and paresed. If the condition be due to trauma or inflammation in the leg or foot, the child refrains from bearing weight by withdrawal of the limb from the ground by acute hip flexion. If, however, the condition be due to paresis, particularly of the quadriceps muscles or the hamstrings or both, the knee will hyperextend if the child can be bribed to attempt to bear all its weight on the limb, or the limb may collapse.

#### *Para-epiphysitis.*

In the pseudo-paralysis of the para-epiphysitis of congenital syphilis the guarding of a limb from activity may mimic poliomyelitis, but the early age, the maternal history, the presence or history of other stigmata such as snuffles, rash, enlarged spleen or a positive Wassermann reaction of the serum of the mother and babe, combined with the finding of definite tender enlargement of the bone adjacent to a joint, will confirm the diagnosis of syphilis.

#### *Pyogenic Infection of Bone or Joint.*

Pyogenic infection of a bone or joint may present difficulties. In a little child a purulent arthritis may be manifested by a "saving of the limb" for several hours before swelling and localized tenderness are evident. In very acute cases of osteomyelitis or periostitis, particularly of the pelvic bones and the shaft of the femur, accurate localization of the tenderness is almost impossible in a very toxic child. These cases may present real problems for immediate diagnosis. The child complains of pain in the limb, will not use it, will not sit up, resents handling, is hurt by rotation of the pelvis and has hamstring tension —all complaints of poliomyelitis patients. A few points are helpful. The degree of toxæmia in poliomyelitis is less, even in the very acute cases, than in osteomyelitis; the cerebration is better. The patient is rarely "cranky," but if sufficient time be given to a gentle examination he will help and cooperate much more than the child with acute osteomyelitis. In both conditions leucocytosis occurs, less pronounced in poliomyelitis, the white cell count ranging between ten and twenty thousand cells per cubic millimetre.

Occasionally when it has not been possible to make a diagnosis, a lumbar puncture has been considered justifiable because of the danger of delay. The absence of cells from the cerebro-spinal fluid excludes poliomyelitis.

#### *Adenitis.*

A child may limp or refuse to use a lower limb to avoid the pain caused by inflammation of the inguinal or deep iliac glands. Careful palpation should disclose this cause.

#### *Rheumatism.*

Rheumatism in older children and young adults may give rise to some suspicion of poliomyelitis, because of fever, pains in the limbs and inability to move. The history of antecedent growing pains, vague ill health or sore throat; the demonstration that no loss of power has occurred, that every muscle group is functioning, but that certain movements, whether passive or active, involving joints, cause pain; the presence of slight swelling around a joint, and the absence of tremor and spine sign would call for the administration of salicylates in effective doses before lumbar puncture would be justified. The children affected by poliomyelitis are usually healthy, sturdy children with no antecedent ill health.

#### *Scurvy.*

Only one case has been seen in which a patient suspected of poliomyelitis had scurvy. The parents brought the baby because he would not move his legs; he would not withdraw them from pin pricks. They were exquisitely tender and hyperæsthetic. When the child was examined the lower limbs seemed almost completely paralysed, yet when the child was supported and induced to stand, the limbs did not give way, the knees did not hyperextend. The discovery of small hæmorrhages in the gums and of red cells in the urine, the history of a diet low in vitamins and the rapid recovery of function after orange juice had been administered for some days confirmed the diagnosis of scurvy.

*Hysteria and Functional Paralysis.*

Fortunately hysteria and functional paralysis are not frequently encountered. Two cases have occurred recently. The first patient was a brother of a paralysed boy; his anxious parents watched him carefully and inquired repeatedly for weakness in the legs. The second was a girl of eleven years who had suffered a mild attack of poliomyelitis in 1928 and recovered after four months' treatment. Two years later, after excessive exercise, thinking the resulting stiffness was incipient paralysis, she became afflicted in the course of an hour with bizarre contractions, acute flexion of knee and hip and an *equinovarus* position of one foot; she maintained this uncomfortable position for days until convinced she was not paralysed. A slight sprain may cause a gross limp in the child of anxious parents. The symptoms in instances of functional disorder do not group themselves in a manner suggestive of poliomyelitis.

*Peripheral Neuritis.*

The association of flaccid paralysis, absent knee jerks and hyperæsthesia in the limbs of an adult may cause confusion between poliomyelitis and peripheral neuritis. The demonstration of definite loss of sensation to touch, heat and cold excludes poliomyelitis, for, although definite changes are found in the posterior root ganglia and hyperæsthesia may be exquisite and persistent, anæsthesia does not occur in poliomyelitis. The history of alcoholism, the presence of Korsakoff's psychosis, the absence of the spinal stiffness or resistance to anterior flexion—a characteristic finding for several weeks after the onset of poliomyelitis in an adult—help to differentiate between the two conditions. The cerebro-spinal fluid in cases of alcoholic neuritis or sciatica differs but little from the normal. Albumin may be slightly increased, but the cells are never above eight per cubic millimetre (Greenfield).

*Lead Poisoning.*

Lead poisoning may be next considered. The differentiation may be difficult in a country where lead poisoning is common. Even in Victoria in six years five instances have been encountered of flaccid paralyses due to lead poisoning. In three of these the paralyses followed repeated convulsions and the diagnosis was further obscured by the appearance of the cerebro-spinal fluid; lymphocytes and globulin are increased in both conditions.

Lead poisoning should be suspected when paralysis follows repeated convulsions. The history of lead colic, of anæmia in members of the patient's family, the demonstration of a blue line on the gums, of basophilic stippling of the red cells and the demonstration of lead in the urine confirm the suspicion. It is well to inquire the occupation of the patient or his parents. In our cases the parents were painters or engaged in glazing tiles, facts which should have warned us.

*Diphtheritic Paralysis.*

Diphtheritic paralysis has not caused difficulty in diagnosis. A few cases of poliomyelitis have occurred within three or four weeks of acute diphtheria, but in these the development of paralysis did not occur at the intervals or in the sequence typical of the sequelæ of diphtheria. Palate weakness occurs in the latter half of the second week of diphtheria, cardiac weakness soon thereafter, paralysis of the lower limbs occurs in the third week and paralysis of the ocular muscles in the fourth and fifth weeks from the initial illness. In diphtheritic paralysis sensory symptoms have not been found constant. Scholes reports diminution of sense of pain to be the commonest, then that of touch, then that of temperature. The absence of hyperæsthesia, of neck stiffness, and of the spine sign, serves to distinguish from poliomyelitis, and examination of the cerebro-spinal fluid gives further assistance, for in diphtheria, while a moderate rise in protein may be found at some stage of the paralysis, it is very rare to find an increase of cells more than 25 per cubic millimetre even at a stage of generalized paralysis.

*Spinal Cord Tumours.*

In the diagnosis of tumours of the spinal cord, if an accurate history can be obtained, the sequence of root pains increasing and radiating more widely, and gradual loss of power during weeks or months, should eliminate poliomyelitis. One case, however, can be recalled in which the incomplete paralysis of flaccid hyperæsthetic lower limbs mimicked poliomyelitis at the first examination. After a period of rest, flexion contractions were observed and these led to the injection of "Lipiodol," by which means the site of compression of the cord was localized.

The presence of yellow cerebro-spinal fluid with excess of protein, but not of cells, indicates either tumour of the spinal cord or polyneuritis. Radiological examination after "Lipiodol" injection distinguishes between them.

*Lesions of Cerebral Vessels.*

The paralysis caused by a lesion of a cerebral vessel such as thrombosis, embolism or hæmorrhage, may be imitated by an upper motor neurone paralysis of poliomyelitis. The patient is middle aged or elderly. The history, the general examination, the blood pressure, enlarged heart or the evidence of cardiac damage, the mode of onset, the absence of fever give information with regard to vascular disease. Pain in the back and neck and between the shoulders, spinal stiffness, limb hyperæsthesia and retention of urine characterize poliomyelitis in the adult.

*Uræmia.*

Even in children we meet cases of convulsions followed by a paralysis, perhaps transient, accompanying the stupor or coma due to uræmia. In several of these patients, suspected of polioencephalitis or tuberculous meningitis, the routine examination of the cerebro-spinal fluid revealed an increase above normal in the quantity of chlorides, which was estimated at 780 or 810 milligrammes per 100 cubic centimetres. This led to further investigations of the renal efficiency, resulting in the detection of gross impairment, as revealed by the high urea content of the blood; later congenital abnormalities or gross kidney disease were found.

*Meningo-vascular Syphilis.*

More confusing may be the sudden development of paralysis of a cranial nerve in the subject of meningo-vascular syphilis.

In the Victorian cases of poliomyelitis a history of diplopia followed by definite paralysis of the third or sixth cranial nerve or complete ophthalmoplegia has not been uncommon. Sometimes paralysis of a cranial nerve has been the only paralysis. In other cases it has accompanied or preceded by several days paralysis of the muscles of the neck or a limb. In all these cases, however, the patient gave a history of fever, headache and pain in the neck before diplopia was noticed, and a spine sign was present when diplopia occurred. A careful history of the remote and recent symptoms, the presence or absence of other stigmata of syphilis usually provide sufficient evidence for differentiation, but examination of the blood and cerebro-spinal fluid for the Wassermann reaction may be necessary. In meningo-vascular syphilis the cerebro-spinal fluid contains an abnormal number of lymphocytes and excess of globulin. These conditions are found in poliomyelitis also, but in this disease the increase in globulin, as shown by the ring with equal parts of saturated ammonium sulphate solution and fluid, is much less pronounced than in syphilis, and polymorphonuclear cells are found along with lymphocytes, their proportion diminishing with the advent of paralysis.

*Tetany.*

It is not difficult to distinguish tetany from poliomyelitis. There is symmetrical spasm of the muscles of the hands and feet—the "carpopedal" spasm. The reflexes are exaggerated and the muscles of the hands and feet may be made to contract by percussing the nerves supplying them (Chevostek's sign).

*Erythrœdema.*

The photophobia, irritability and atonia of erythrœdema (Swift's disease) may be confused with or regarded as sequelæ of polioencephalitis. Once seen, however, the syndrome is remembered. The care-worn, weary mother,

the irritable, peevish infant hiding from the light with flexed head, the subaminal transient rashes on the trunk and limbs, the red swollen and later desquamating palms and soles, the atonia of the muscles, and, in some cases, the ulcerating mouth and gums, present a clear-cut picture. Experimental work may yet shown some ætiological relationship between the two conditions, but at present it is not difficult to recognize the clinical entity of Swift's disease.

### Poliomyelitis Simulated by Acute Febrile Diseases.

The second division of Group I, the acute febrile diseases which may be imitated by the pre-paralytic stage of poliomyelitis, will be next considered.

#### *Malaria.*

In countries where malaria occurs the natives, accustomed to its fever, backache and headache, tend to regard as similar the acute stage of poliomyelitis and rarely complain until paralysis has occurred. In Rabaul the severe pain between the shoulder blades was found helpful in detecting the disease in sufferers who sought treatment early.

#### *Tonsillitis.*

Suspicion of poliomyelitis may arise from tonsillitis, especially when it is accompanied by swelling of the posterior cervical glands, making movement of the neck painful. In poliomyelitis some faucial engorgement is usual; the children affected frequently have large tonsils, but actual follicular tonsillitis or exudate rarely occurs. Distinction can be made, however, between these conditions and poliomyelitis by the fact that, whereas in the latter the spine sign is elicited by pain on anterior flexion only, in those cases in which neck rigidity is due to tender cervical glands or to myositis, movements of extension and lateral bending are as painful as flexion. One should be careful to ascertain, before deciding that a spine sign is absent, that no paralysis of the extensor muscles of the neck has occurred. The absence of popliteal pain, of tension in the hamstrings, of pain in rotating the lower part of the spine anteriorly, usually enable one to exclude poliomyelitis without resort to examination of the cerebro-spinal fluid.

#### *Otitis Media.*

*Otitis media*, particularly in infants and little children, may confuse. The fever, irritability when disturbed, drowsiness and vomiting are common to patients suffering from both conditions. Before lumbar puncture is carried out in a patient suspected of poliomyelitis because of fever and a stiff neck, the ear drums should be examined. Two cases have been seen in which facial nerve palsy accompanied *otitis media* of the same side.

#### *Pyelitis.*

As well as the routine examination of the *membrana tympani*, the routine microscopical examination of the urine for pus cells and bacilli is a wise procedure before one considers the performance of lumbar puncture in a feverish, irritable infant or little child who resents anterior bending. Pyelitis may be accompanied by drowsiness.

#### *Sunstroke.*

The high fever following sunstroke may cause complaint of pain in the neck, but not enough to suggest a positive spine sign. The patient's body can be passively flexed.

In an acute febrile condition, which may or may not be the onset of poliomyelitis, the patient has to be kept under close observation and lumbar puncture postponed until a definite spine sign is present. The administration of fluids very freely, of glucose, of an aperient, may be followed by improvement and by diminution of the degree of neck stiffness, which make one hesitate about the decision to perform lumbar puncture.

#### *Dysentery.*

During January and February, when epidemics of bacillary dysentery and poliomyelitis synchronize, the association of fever, drowsiness, irritability and hyperæsthesia has given rise to a suspicion of poliomyelitis, but the passage of the typical muco-purulent motions has shown the cause to be intestinal.

#### *Pneumonia.*

Of all the problems for differential diagnosis patients suffering from pneumonia with meningismus cause the greatest anxiety at the first examination. The fact that in children signs in the chest are often delayed, may steal from us the help given by the use of the stethoscope and percussion. One hesitates to carry out lumbar puncture on a child who may have pneumococcal septicæmia, yet if a definite spine sign be present, a delay of twelve hours may result in widespread paralysis. To complicate the problem further, in some epidemics the systemic phase of poliomyelitis may be accompanied by involvement of the respiratory system with definite pneumonic signs. Fortunately in Melbourne only one case of this type has been encountered.

Fever, rapid pulse, drowsiness and flushed appearance are common to both conditions. One is driven to a tentative diagnosis based on many little points; grunting respirations, a short cough, slight contraction of the *alæ nasi* weigh in favour of pneumococcal infection. Tremor, slight photophobia, delay in emptying the bladder, hyperæsthesia of a limb, point to poliomyelitis. As a rule, the child with pneumonia is more toxic, less inclined to talk, is drowsy while being examined, and cooperates less and is less interested than the child with poliomyelitis who, if satisfied you will be gentle and will not hurt him, will talk about his home or school and answer questions.

Slight diminution in breath sounds at the base or apex of a lung and slight impairment of percussion may justify one in waiting a few hours when definite signs may be present. The use of the fluoroscopic screen has been found of value in this type of case at the Children's Hospital, Melbourne, in revealing consolidation before we could detect it clinically. When an epidemic of poliomyelitis is at its height, with higher temperatures, greater toxæmia than in the early weeks, it may not be possible to differentiate at the first examination and delay of more than a few hours for a second examination, when the child has more confidence, may not be justifiable.

Before lumbar puncture is performed, provision should be made for bedside examination of the fluid, and the amount of fluid withdrawn should not exceed two cubic centimetres, a quantity sufficient for examination for cells and the test for globulin by means of ammonium sulphate.

#### *Tetanus.*

Occasionally one may have real difficulty in distinguishing between the two emergencies, acute tetanus and pre-paralytic poliomyelitis. In the latter the spinal stiffness may sometimes develop rapidly to the degree of opisthotonos typical of tetanus. In tetanus, fever, headache and pains in the back are present, and a history may be given of a wound some days before. In two such cases seen at the Children's Hospital, the absence of *risus sardonicus*, of trismus, of masseter rigidity and of tonic spasm enabled us to diagnose poliomyelitis, although we lost, through blood contamination, the help of cerebro-spinal fluid examination. No changes have been detected in the cerebro-spinal fluid of tetanus (Levinson).

We need not discuss, in Australia, the diagnosis from rabies in which salivation, convulsions and difficulty in swallowing due to pharyngeal spasm may cause confusion with difficulty in swallowing due to cervical rigidity and pain on flexing the head to bring the cup to the mouth.

#### *The Exanthemata.*

The skin rashes which may accompany poliomyelitis may simulate the eruption of any one of the acute exanthemata. A fine petechial rash, a measly rash with blotches and a scarlatinal blush have all been noted. Little difficulty arises in distinguishing these cases from measles, German measles or scarlet fever. The absence of coryza and Köplik's spots, the early appearance of the blotchy rash on the trunk and extremities distinguish poliomyelitis from measles, in which the rash appears first on the face after three days of illness. The scarlatinal rash of poliomyelitis usually appears later, when the fever is declining and when other signs are available for differentiation. Desquamation may be pronounced and continued for many weeks.

**Diagnosis Aided by Examination of the Cerebro-Spinal Fluid.**

Group II, in which examination of the cerebro-spinal fluid is required for purposes of differentiation, will now receive consideration. Cases which fall into this group are more straightforward than those of Group I, division (*b*), in which the diagnosis depends on clinical experience. In this group examination of the cerebro-spinal fluid enables one to prove the diagnosis.

We need not dwell on differential points in the clinical examination. The acute onset with fever, delirium, headache and the evidence of increased intracranial pressure, the stiff back and retracted head or bulging fontanelle indicate the necessity for lumbar puncture. The appearance of turbid or purulent cerebro-spinal fluid reduces the number of possible ætiological agents. Examination of a film stained by Gram's method and culture further narrow the possibilities.

*Meningitis.*

*Cerebro-spinal Meningitis.*—In cerebro-spinal meningitis at a very early stage the fluid may be clear or slightly olive, and it may be under great pressure. A coagulum may form if the vessel containing the fluid be allowed to stand for a time. More frequently there is slight turbidity and the numbers of cells are enormously increased; there may be 2,000 per cubic millimetre, almost all being of the polymorphonuclear type. A careful search of a film stained by Gram's method is usually rewarded by the detection of a few leucocytes containing several pairs of the characteristic kidney-shaped diplococci of Weichelbaum which do not retain Gram's stain; or the organisms may be found between the cells. The protein in the cerebro-spinal fluid is greater in quantity than the normal, the chlorides are diminished in amount and glucose is almost absent. The diagnosis, however, depends on the discovery of the typical organism.

*Pneumococcal and Streptococcal Meningitis.*—The forms of meningitis due to the pneumococcus and streptococcus may be considered together. The intrathecal pressure is increased, the cerebro-spinal fluid has a flocculent coagulum and is turbid, the protein content is higher and the content of glucose and the chlorides lower than normal. The cells are polymorphonuclear and the causative organisms (Gram positive) can be seen lying between them. By culture further differentiation can be made. Cases of pneumococcal meningitis have been recorded in which the cellular reaction was slight and the organisms so numerous as to give a diffuse turbidity to the fluid. These cases emphasize the necessity for examining a film stained by Gram's method before venturing an opinion on any opalescent cerebro-spinal fluid.

*Staphylococcal Meningitis.*—Staphylococcal meningitis occurring as a complication of septicæmia, in pyæmia, or secondary to an empyema of a cranial air sinus, is diagnosed by the discovery of the cocci in the film and by culture. In other respects the fluid resembles that of streptococcal meningitis.

*Influenzal Meningitis.*—In the spring of 1930 a small series of cases of influenzal meningitis occurred in Melbourne. The mortality was 100%, the oldest patient affected two and a half years of age. The onset was acute, the fever very high, the respirations rapid; drowsiness, irritability, repeated convulsions, head retraction and bulging fontanelle were constant signs, which in some instances led to the suspicion of polioencephalitis; but the cerebro-spinal fluid was purulent and the polymorphonuclear cells contained and were surrounded by incredible numbers of bacilli which did not stain by Gram's method. Culture on blood media resulted in identification of the bacillus. Rivers states that the presence of indol in the cerebro-spinal fluid is pathognomonic of influenzal meningitis.

*Tuberculous Meningitis.*—Of the conditions classified under Group II the disease which presents most difficulty in differential diagnosis from pre-paralytic or early poliomyelitis is tuberculous meningitis. The age incidence is very similar: fever, headache, drowsiness, irritability, neck stiffness and spinal rigidity are common to both conditions. In both conditions the cerebro-spinal fluid is clear, under increased pressure and contains excess of cells and globulin.

If a patient be brought for treatment early, diagnosis is essential, because, with the suspicion of poliomyelitis hanging over us, we dare not "wait and see" what happens—whether tubercles will develop in the chorioid or the spleen will enlarge, or the condition progress to coma or a fatal conclusion.

The history is most important. Usually if one questions the mother of a child with tuberculous meningitis, she will remember that he has been "off colour," peevish, tiring easily, coming in from play to lie down, wakeful at night, and not eating well for weeks or even months. Occasional vomiting, constipation and abdominal distension may have been observed, contrasting with the history of sturdy health which is the usual story of the child who develops poliomyelitis within a few days or, at most, two weeks of a short, vague illness. The history of an attack of measles or pertussis some months earlier or the visit of a tuberculous relative may be remembered if asked for, but is rarely volunteered.

As a rule the child with tuberculous meningitis is much more difficult to examine than a child the same age and temperament suffering from early poliomyelitis; he is cranky, will not cooperate, turns over abruptly and puts his face to the wall in the middle of a test—a marked contrast to the vivid alertness of the child suffering from poliomyelitis even in a most severe form. Enlargement of the cervical or hilum glands or palpable enlargement of the abdominal glands and enlarged spleen point to the existence of tuberculosis. In both diseases hydrocephalus may develop with bulging of the fontanelles; palsies of cranial nerves occur in both conditions. The local palsies of the face and limbs in tuberculous meningitis are usually transient or shifting—alterations which do not occur in poliomyelitis. Convulsions and twitchings are much more commonly found in tuberculous meningitis, for repeated convulsions are so uncommon as heralds of poliomyelitis that when they occur it is wise to seek for another diagnosis.

The cerebro-spinal fluid in tuberculous meningitis is under greatly increased pressure, usually clear or colourless, but may later have a yellowish discoloration. A fibrin web frequently forms if the fluid is not shaken and in this or in the deposit obtained from prolonged centrifugalization, bacilli may be found in films stained by the Ziehl-Neilsen method. The cells are increased in number; usually there are between 100 and 300 per cubic millimetre, but counts up to 1,000 may occur. The numbers of both polymorphonuclear and mononuclear cells are increased, the ratio varying in different patients and at different stages of the illness of the same patient. So we obtain no help there in discriminating between the two conditions. Globulin and protein are increased in quantity, but the content of chlorides is constantly reduced in a progressive manner. In the earlier stages, readings between 680 and 700 may be obtained, but within a week the quantities may have fallen to 600 or 650 and, later, even to 550 milligrammes per 100 cubic centimetres.

The estimation of the amount of chlorides has been found most valuable in distinguishing between the two conditions. In this connexion a simple but important point to be borne in mind is that the fluid must be dropped from the needle into a dry test tube. The chlorides in the cerebro-spinal fluid of a patient suffering from poliomyelitis are not reduced in amount at any stage of the disease, whereas, whenever real difficulty has been encountered in differentiating the two conditions, the course of the disease has confirmed the diagnosis of tuberculous meningitis based on the estimation of the chlorides, even when readings which might have been regarded as equivocal, for example, 690 milligrammes per 100 cubic centimetres were obtained. The test is very easy and sensitive.

A fine web of coagulum is not uncommon in poliomyelitis, but it is found more often after the development of paralysis than before, and thus is not of diagnostic value. The estimation of the chlorides can easily be carried out at the bedside along with the cell count and globulin estimation.

*Typhoid Fever.*

The high temperature, delirium, tremor, epistaxis, twitching and severe headache in a severe case of polio-

myelitis may be diagnosed as due to typhoid fever, but the comparatively rapid onset, the rapid pulse, the presence of a spine sign, the absence of rose spots or leucopenia and the enlargement of the spleen usually make the diagnosis clear. More difficulty has been experienced with the cases in which a meningitis due to the *Bacillus typhosus* occurs as a complication of typhoid fever, until one obtains a clear history of the onset, and proper investigations have been carried out; in this condition there are neck stiffness and a spine sign.

The cerebro-spinal fluid in typhoid meningitis is clear or turbid, rarely purulent; the cells may number as many as 100 per cubic millimetre. Lymphocytes predominate in the milder cases, polymorphonuclear cells in severer cases. The bacillus can be seen microscopically and cultivated from the fluid which reacts to the Widal test. In typhoid fever the quantity of chlorides of the cerebro-spinal fluid falls early in the disease and remains low until convalescence is well established.

*Parotitis.*

The rare nervous sequelæ of epidemic parotitis may cause a tentative diagnosis of polioencephalitis or poliomyelitis to be made. It is said that meningeal symptoms may occur when there is no other evidence of the disease. In one case a little brother had suffered from mumps three weeks earlier; in addition the patient had swollen, tender parotid glands, so that the diagnosis was made easy; examination of the cerebro-spinal fluid revealed the changes described in text books, namely, clear or shimmering fluid, excess of cells which may number over 2,000 per cubic millimetre, and are almost all mononuclear, and a slight increase in the content of globulin and normal chlorides. Routine examination of the cerebro-spinal fluid of young adults affected during epidemics of parotitis has revealed some lymphocytic increase to be common, even when no meningeal signs are present.

*Vaccination and Varicella.*

The scars of recent vaccination or of healing pocks establish the diagnosis in the cases of encephalitis which may follow vaccination or varicella. These present many interesting ætiological problems, but none in differential diagnosis.

*Subdural or Subarachnoid Hæmorrhage.*

The symptoms following subdural or subarachnoid hæmorrhage may cause confusion with polioencephalitis or poliomyelitis. The hæmorrhage may be spontaneous or result from a trivial injury, the patient recovering from its immediate effect and thereafter developing headache, drowsiness or lethargy, perhaps delirium, with alterations in the reflexes.

Subarachnoid hæmorrhage may be accompanied by neck stiffness and pyrexia, subdural hæmorrhage by convulsions, hemiplegia, vomiting and perhaps papillœdema. The changes in the cerebro-spinal fluid due to subdural hæmorrhage are slight and equivocal—little more than a yellowish discoloration. Subarachnoid hæmorrhage, however, causes the fluid to be evenly blood-stained. The use of Sicard's test is the most trustworthy method of distinguishing between this condition and discoloration by blood obtained from the accidental puncture of a vein within the theca. A yellow coloration of the clear supernatant fluid after centrifugalization or sedimentation is always an indication of preexisting subarachnoid hæmorrhage; the depth of colour varies according to the length of time which has elapsed between the hæmorrhage and the withdrawal of the fluid.

Recently a child of three years was brought to hospital two days after a fall of 90 centimetres (three feet). He had vomited, then appeared to have recovered, but head retraction was noticed the following day; this gradually increased, and he complained of pain in the neck. When examined he lay with his head retracted. Any attempt to move the head from the pillow or in any direction was resented and the cerebro-spinal fluid contained blood. Although he was seen during an epidemic of poliomyelitis, it was possible to exclude this by the absence of fever or tremor, by the restriction of the pain to the cervical region, by the absence of hamstring tension and of any resistance to flexion of the lumbar spine, and by the fact that movement of the head in any direction whatever was apparently equally painful.

*Encephalitis Lethargica.*

In conclusion the differentiation of polioencephalomyelitis from *encephalitis lethargica* will be considered. In Australia epidemics of the latter occurred in 1919 and 1924; poliomyelitis has been more frequent since 1925. This may explain the fact that while, academically, great confusion exists between the two diseases, in actual practice, there have been very few instances of suspected polioencephalitis which have been proved to be *encephalitis lethargica*. Unfortunately, the converse does not hold. Only too often an acute illness with fever, drowsiness and involvement of ocular or facial muscles in young adults has been regarded as *encephalitis lethargica*. Despite tremor, spine sign, hyperæsthesia and retention of urine it has been allowed to progress to widespread limb paralyses without any attempt having been made at treatment. While admitting that *encephalitis lethargica* may occur sporadically, while appreciating the interesting ætiological problems it presents, one cannot help being impressed by the tragic confusion of a disease for which we have no means of therapy, with one for which a specific therapy is available. Poliomyelitis is common in Australia, *encephalitis lethargica* at present is rare. Paralyses of muscles of the eye, face and palate are common occurrences and often comparatively early signs in poliomyelitis. This tragic confusion will be avoided if we eliminate the notion that an acute illness accompanied by drowsiness and irritability, followed by paralysis occurring above the collar, can be labelled *encephalitis lethargica* and left at that.

The distinction between the two diseases (the encephalitis described first by Economo in 1917, and polioencephalomyelitis known for many years before) lies in the nature of the action of the virus on the nerve elements. In the latter there is a sudden sharp attack which develops rapidly and in a few days attains the maximum of severity, quickly producing the greatest extent of destruction. In epidemic encephalitis the onset is frequently very gradual, the progress of the disease slow, in some instances requiring several months to reach its greatest severity, and even then the findings have a tendency to variability, being subject to exacerbations and remissions.

The serum of patients convalescent from encephalitis has no immunizing power against poliomyelitis virus. Poliomyelitis can be transmitted to monkeys with a fair degree of ease, whilst most attempts to transmit epidemic encephalitis to animals have failed. The two diseases differ histologically.

These points, however, do not assist in any bedside diagnosis of the condition of a patient acutely ill. Greenfield and Carmichael, in their invaluable book, "Cerebro-Spinal Fluid in Clinical Diagnosis," summarized the fluid findings in *encephalitis lethargica* as follows:

1. The pressure is usually raised, the appearance almost always water-clear, though occasionally blood admixture occurs from small meningeal hæmorrhages.

2. No coagulum ever forms, except when there is meningeal hæmorrhage. Colourless fluids never show the slightest fibrin coagulum, a point of distinction from poliomyelitis in which the formation of a fine fibrin web is not uncommon.

3. A slight or moderate lymphocytic increase is almost always present during the early weeks of the disease; the count rarely exceeds 100 cells per cubic millimetre. It is always a pure increase of mononuclear cells, except when there is hæmorrhage. This is a second important point in diagnosis from poliomyelitis, in which polymorphonuclear cells are most increased in the early stages and are present up to the first week after paralysis.

Estimation of the glucose or protein content does not assist in the differential diagnosis. The diagnosis of *encephalitis lethargica* must rest upon observation of the patient over several weeks when the gradual progress, the remissions, the development of mental changes or alterations in the disposition provide further differentiation from polioencephalitis.

In conclusion, in the diagnosis of poliomyelitis before the onset of paralysis, three practical points should be remembered:

1. The fluid should fall from the needle into a dry test tube.

2. The examination for cells should be carried out without delay. Lyons has shown that cytolysis occurs in the cerebro-spinal fluid after withdrawal and this is selective; the polymorphonuclear cells are destroyed more rapidly than the mononuclear.

3. The variation in the fluid at different stages of the disease is pathognomonic of poliomyelitis. The polymorphonuclear cells are increased early in the preparalytic stage, but quickly disappear as the lymphocytes increase, until at the stage of paralysis only 5% or 10% of cells are polymorphonuclear. Globulin, while present in the preparalytic stage, does not reach its highest concentration until paralysis has developed and the cell count of the cerebro-spinal fluid is diminishing.

JEAN MACNAMARA, M.D., B.S. (Melbourne),
*Honorary Medical Officer to the Physiotherapy Department, Children's Hospital, Melbourne; Consultant to the Victorian Committee for Poliomyelitis.*

# British Medical Association News.

## SCIENTIFIC.

A MEETING OF THE OPHTHALMOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Eye and Ear Hospital on September 2, 1930, DR. MARK GARDNER, the President, in the chair.

### Ophthalmological Aspects of Psychiatry.

DR. REG. ELLERY read a paper entitled "Ophthalmological Aspects of Psychiatry" (see page 368).

DR. F. J. B. MILLER asked whether Dr. Ellery agreed that thorough ophthalmological examination should be made of insane persons.

DR. J. F. SPRING advanced the opinion that there was a tendency for the ophthalmologist in his general hospital work to regard as ophthalmologically normal any patient in whose eyes there was no obvious abnormality. He made mention of a patient who had received exhaustive and skilled treatment for renal glycosuria. Eventually, when the fields of vision had been carefully mapped out, the existence of an incomplete bitemporal hemianopia had been found; this had led to the diagnosis of a tumour in the pituitary region.

DR. W. J. L. DUNCAN asked if Dr. Ellery had been able to satisfy himself that the appearance of the optic disc of a person suffering from *tabes dorsalis* differed from that of a patient suffering from general paralysis.

DR. MARK GARDNER said that he had nothing to add to the discussion, but wished, in the name of members, to thank Dr. Ellery for his paper.

Dr. Ellery in reply to Dr. Miller stated that he thought the ophthalmological examination of persons certified as insane could well be made more thorough. In 15% of patients suffering from disseminated sclerosis the mental symptoms so dominated the picture that it was very easy for such patients to be wrongfully certified as insane. That was just one of the many lesions in which thorough ophthalmological investigation would minimize the risk of error. He would like to see a closer cooperation yet between the neurologist, the psychiatrist and the ophthalmologist.

In reply to Dr. Duncan, Dr. Ellery said that he had never attempted to differentiate between the appearances of the optic disc in persons affected with *tabes dorsalis* and general paralysis. He felt the tendency was to regard neuro-syphilis as a general disease which could not easily be divided into distinct clinical entities as was suggested when general paralysis and *tabes dorsalis* were spoken of as distinct from one another. He preferred a classification which was determined by the presence or absence of symptoms which were known to have a bearing on prognosis.

### Demonstration of Patients.

DR. A. S. ANDERSON showed a boy, aged nine years, whose left eye had been watery since birth. No puncta were visible. If no canaliculus had been found at operation, Dr. Anderson had intended to insert a cannula from the conjunctival sac to the lachrymal sac, after the technique of Antili Pockley. However, at operation a well developed canaliculus was found and Lister's three snip operation was sufficient to produce a cure of the condition.

Dr. Anderson's second patient was a boy, aged six years, who had reported first in July, 1930. Two years previously he had suffered from meningo-encephalitis. He had been affected with diabetes for nine months. During the previous few weeks he had complained of headaches and failing vision. Examination of the fundi revealed evidence of old retinitis. There was slight papillœdema on the right side. The fields were full and the vision reduced to counting fingers at 60 centimetres (two feet) in the right eye and 120 centimetres (four feet) in the left eye. He was able to appreciate colour in very large objects only. Radiological examination of the skull revealed no abnormality. There was no reaction to the Wassermann test. The papillœdema subsided and well-marked temporal pallor was observed on both discs. In spite of this his vision improved until on October 16, 1930, he was able, by using the right eye only, to count fingers at six metres (twenty feet), and could read $^{6}/_{12}$ with the left eye. He was then able to recognize the colour of objects two millimetres in diameter.

DR. ESME ANDERSON showed a man who had been sent to hospital with a history of injury to the right eye sustained while hammering. There was a mass in the vitreus, there was no red reflex and X ray examination failed to reveal an intraocular foreign body. Milk injections had been given; the eye had quietened down and the tension was normal. The question arose as to whether it was worth preserving the eye for cosmetic reasons or advisable to remove it.

The opinion of the members present was that the injury had had nothing to do with the condition and that the eye would probably shrink.

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A MEETING OF THE OPHTHALMOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Eye and Ear Hospital, Melbourne, on October 31, 1930.

### A Visit to Canada.

SIR JAMES BARRETT gave a short account of his recent visit to Canada and made full acknowledgement of the kindly and helpful assistance given by the members of the medical profession in Canada. He had an opportunity of meeting the ophthalmic and aural specialists in a body at Vancouver and individually in other cities. Three lectures on bush nursing were given in Canada, namely, at Vancouver, Winnipeg and Edmonton.

In Western Canada there were two complete medical schools, namely, in the University of Manitoba, at Winnipeg, and the University of Alberta, at Edmonton. In both instances the associated hospital was managed, as was the practice in Canada, by a medical superintendent under a board of management. This officer did not practise in or out of the hospital, but was simply a medical administrator. The hospital at Edmonton was closely associated with the university and adjoined the medical school of the university. The university appointed half the members of the board of management. Both schools appeared to be highly organized.

In the universities of British Columbia and Saskatchewan, only the preliminary part of the medical course could be undertaken at present. In both the complete medical schools the number of medical students was limited notwithstanding the fact that they were State universities in the same sense that the universities of Sydney and Mel-

bourne were State universities. There was room for only so many students, and the university exercised discrimination and rejected the number in excess of the places available after investigation of the character of the would-be students.

The ophthalmic work in all these cities seemed to him to be well organized and conducted at a high level of efficiency. The meeting of the Ophthalmic Section of the British Medical Association at Winnipeg was especially interesting. A detailed account was given by first hand observers of the latest methods adopted for the treatment of the detachment of the retina. The verdict seemed to be that in a certain minority of cases the method was effective. Dr. Royle's paper on the operative treatment of *retinitis pigmentosa* caused much private discussion. The problem of the examination of those on duty during marine accidents on the Australian coast was referred to London to the Central Body of the British Medical Association for consideration and report. The definition of blindness as adopted by the Charities Board of Victoria, on the recommendation of the Australasian Medical Congress of 1923, was also referred to London for consideration with the hope that uniformity might be obtained.

The section dealing with medical sociology in the history of medicine, which had just been established, held two successful sessions in which the immigration problem was dealt with at length; a number of recommendations were made to the central body. It was unanimously agreed that the history of medicine should form a subject for examination for all the higher degrees.

A hearty vote of thanks was passed to Sir James Barrett for his very interesting and informative address.

### Demonstration of Patients.

Patients affected with disease of the eye were shown by Dr. Mark Gardner, Dr. Gault, Dr. A. S. Anderson, Dr. Esme Anderson and Dr. Max Yuille.

### Pathological Library.

It was decided to institute a pathological library.

## Obituary.

### CHARLES HENRY MOLLOY.

Dr. Charles Henry Molloy, whose death, as announced in a previous issue, occurred on January 21, 1931, was born at Ballarat, Victoria, on July 11, 1863. He was the second son of the late James Molloy, who came to Australia by sailing ship in 1857. James Molloy married Sarah Neale who had travelled with him from Ireland, and their second son, Charles, was born at Ballarat. Here the boy went to Saint Paul's Church School, where his father was headmaster. His mother was a woman of strong character and from her he inherited many of the qualities which distinguished him in later years. After spending some time at Ballarat College, Charles Molloy went to the Church of England Grammar School Melbourne. Here he came under the influence of Professor E. E. Morris. Somewhat shy and retiring in disposition, Molloy still had sufficient independence of character to enable him to hold his own among the other boys and to pursue his studies with quiet enthusiasm. He won many school prizes.

In 1881 Molloy became a medical student at the University of Melbourne and five years later he graduated as Bachelor of Medicine. In 1892 he took the degree of Doctor of Medicine. After graduation he obtained at Beechworth an appointment as resident medical officer. He also became resident medical officer at the Melbourne Hospital and remained there until April, 1887. He then went to the Alfred Hospital, where he held both junior and senior resident appointments. After serving during 1888 and 1889 as Officer in Charge of the Municipal Fever Camp at the Alfred Hospital, he returned to the Melbourne Hospital as Medical Superintendent. He served in this capacity for ten years and gained experience which helped him considerably in later years. He left Melbourne in 1900 for Tarwin, South Gippsland, intending to rest and recruit his health; he stayed, however, and built up a large practice. His advice and help were sought by people scattered over a large area and he strove to help them. The last five years of his life were spent in Meeniyan. Here his life was less strenuous and after a little more than two years of failing health, borne without complaint, he died suddenly while walking in his garden.

Charles Molloy was a member of the British Medical Association in his early years. He was a member of the Council of the Victorian Branch in 1892 and served for two years. He severed his connexion with the Association for a few years and was later on reelected as a member. He was a student and fond of reading, which formed his chief recreation. He was a man of character who did what he could to improve the lot of his fellow man.

Dr. F. A. Wood writes:

My real knowledge of the late Dr. Charles Molloy came when I settled in South Gippsland in the late months of 1915. Dr. Molloy had at that time relinquished his widespread practice to perform military duty, first at the Seymour Camp and later on board a troopship to Egypt.

I had undertaken to try and do as much work for him as was possible in his absence (which I esteemed a privilege), and on his return in 1916 there commenced a period of fourteen years when we were very much thrown together in our work; and during this long stretch I learned how great a privilege it was to have such a loyal and honourable colleague, possessed of great ideals of work, and one who always brought light and aid in every difficult situation.

Dr. Molloy's work amongst his patients was characterized by great thoroughness and by abounding kindness and thoughtfulness for them. No journey on the rough roads of South Gippsland was too long and no detention too great, if by any means he could alleviate suffering; and he frequently played the part of doctor and nurse for many hours until the patient could be safely left in good hands. In fact, self-sacrifice was the most marked feature of his work apart from the splendid powers of diagnosis and treatment he always had at his command.

He came to South Gippsland to recruit his health, as he possessed a property here, and the people of the bush rapidly learnt to rely on him and to know that they possessed in him a medico who gave of his very best.

He was a man whose one relaxation was reading, and it was remarkable how up to date he remained.

During the last two years his health had failed, and he began to realize that he could not work so hard, but he persevered until the end came with very great suddenness, and we all realized what a tower of strength had been removed from us.

The funeral, of the simplest character in accordance with his wishes, took place at Meeniyan on January 23 and brought together a large band of mourners who, one and all, realized the worth of the doctor and friend who had passed on and left them so much the poorer.

Dr. A. V. M. Anderson writes:

Dr. C. H. Molloy became Resident Medical Officer to the Melbourne Hospital in October, 1886, and made such use of his opportunities there that he was selected to fill a similar position at the Alfred Hospital in June of the following year.

When I went to the Alfred Hospital in March, 1888, I began an association with him which was intimate for many years and which lasted for the remainder of his life.

He became Medical Superintendent of the Melbourne Hospital in July, 1889, and occupied that position for ten years. It was a matter of surprise to many that he did not then embark on the career of a consulting physician in Melbourne. His experience and knowledge were great and he had a large circle of acquaintances among medical men in Melbourne. He had a very retentive memory and was extremely observant—in fact, I know of no one who had cultivated his faculties of sight, hearing and touch to as great a degree as he had.

But he was reserved and unconventional, and disliked all kinds of formality, ceremony and publicity. While at

the Alfred Hospital, he had decided that he would live ultimately in the country; to this end he had begun negotiations for the purchase of a property in Gippsland and here he took up his residence on leaving Melbourne. While not desirous of leading a busy professional life, his gifts and attainments soon became known and his services were much in request, especially as a consultant in cases where his experience and sound common sense made his opinion very valuable.

His interests were wide; he was a discriminating reader of good literature and was fond of sport; he was frequently to be seen in Melbourne during test cricket matches and retained his club membership.

He was on military duty for a time during the Great War and suffered considerably from climatic conditions in Egypt. His health after his return was not so good as previously and a serious illness in 1928 incapacitated him for a time. But he was able to resume practice and continued working, as he would have wished, up till the last day of his life.

It is a fortunate thing that men of his mental and professional calibre are not uncommonly found in country districts and one feels that his loss will be deeply regretted by his patients and Gippsland *confrères*, as well as by those who enjoyed the privilege of his friendship in Melbourne.

## GEOFFREY FREDERICK TRAVERS.

DR. GEOFFREY FREDERICK TRAVERS, whose death on February 1, 1931, has already been recorded in these pages, was born in Sydney in 1857. He was the son of James Lindsay Travers, sometime tea merchant of London. When his father died, Geoffrey Frederick Travers was sent to England to the care of relatives. He was brought up at The Priory, Warwick, and was sent to school at Clifton College. He studied medicine at Saint Bartholomew's Hospital and in 1883 gained the diploma of membership of the Royal College of Surgeons and that of licentiate of the Royal College of Physicians. In the following year he returned to Australia and started practice at Prahran. He remained there for seven years and returned to England. In 1893 he came back to Victoria and practised in Gippsland until 1900, when he removed to Elsternwick. He remained at Elsternwick until he retired from active practice for health reasons. He is survived by his widow, one son and a daughter.

# Correspondence.

## THE WAR AND SIR NEVILLE HOWSE'S PART THEREIN.

SIR: Since the publication of my letter last Saturday, March 14, 1931, the editor of "The Medical History" has very courteously written me to the effect that the statement "that Sir Neville Howse was one of the best directors of medical services at the war" does not appear in "The History," nor words that can be paraphrased as such. He suggests that, in justice to himself, the impression be corrected in the journal. To this I gladly accede, with the apology that the belief was brought about by the use of inverted commas and the context, the discussion apparently being centred on the truth or otherwise of this statement.

At the same time, without modifying in any way the purpose of this communication, may I say that by commendation of one director of medical services and depreciation of others—Generals Willams, Ford, Birrell—the sense of the statement is by implication well emphasized in "The History."

This being so, I feel justified in saying that, though the thread of my argument, which was to show that Dr. Springthorpe's letter pointed certain lessons we were failing to recognize, may be disturbed, the substance of it remains intact.

In amplification of the above and in exoneration of myself, I ask you to publish again the following paragraph, the final one in my previous letter:

> In conclusion, may I say, in the event of any wrong inference in this letter having been drawn as to the plan and form of Colonel Butler's work, that since reading the controversy—on account of absence from home, the several copies of the journal reaching me at the one time—opportunity has not permitted of the purchase and the reading of the book. The inferences drawn are from the statement itself, the knowledge that "The Official History of the War" was being used as a book of reference in the preparation of this history and the allusions in Dr. Springthorpe's letter to praise and blame and future lessons.

Yours, etc.,

H. E. JACKSON.

Bendigo, Victoria,
March 17, 1931.

SIR: Permit me to finalize my share in this controversy:

1. As regards material medical facts, up to March, 1916, not one has been controverted; and, comparing wherever comparable, Anzac was surely little, if any, better off than the Crimea, or our 3 A.G.H. nurses at Lemnos than the "Lady of the Lamp" at Sentari—sixty years previously.

2. As regards consequential opinions, "*tot homines, tot sententiæ*." The historian and I, looking from different angles, differed here and there in our views and we agree to differ until the final review. My article of January 31, 1931, arose, however, from none of these, but from the eulogium in yours of December 6, that the subject of controversy "was one of the best D.M.S. of the war." My experience was to the contrary, as I showed constructively, and my three years' subsequent experience, under greater issues and greater tests, confirms my view.

3. A staff officer's triple quotation of Sir C. B. White's views deserves respect. But they were non-medical and do not contradict one fact. In 1919 "C.B." said to me: "I am afraid you were expecting miracles." "No," I replied, "but common sense," and my article explained the failure. "C.B." takes his place amongst the immortals for his almost unique success. Did I ask him: "Was the evacuation a miracle"? No, because it was the deserved result of superlative ability against almost impossible odds.

I thank Colonel Butler for his characteristically firm but courteous comment and warmly support his two suggestions: (*a*) "defer judgement" and (*b*) "beg, borrow or buy" his Volume I, also his Volume II when it appears. But why not also glance through my own little diary, copies being available in private and official hands, in the Mitchell Library and in due course in our own Medical Library.

I also thank Dr. R. M. O. Jackson. But unfortunately history cannot be confined to matters of fact; it must take in also questions of fallibility and negligence. And, in my opinion, the future is far more important than the past and due weight must always be given to the rank and file.

My own right to criticize having been established and the historian's own adverse criticism of two distinguished surgeon-generals duly noted, I should have ended here, but for the continued deluge of animadversion which has been self-revealing rather than warranted, because not applicable and not true. On looking into the wonderful index, I place my critics as follows. Two are not mentioned, both senior officers, one not on the staff and never in personal contact with the departed, but the other an A.D.M.S. "who knows," but "wonders if I thought Howse was the Almighty." I wonder if he ever read the medical version of the Athanasian creed. Six others were from field ambulances (H.'s high water mark) and became A.D.M.S.—all enjoyed promotion and practically all rose to be staff officers. I mention only the findings of one (though almost all were extraordinary), the critic who questioned my real rank, and that only because it enables me to quote from an appreciative letter of Sir H. B. Allen (Dean of our Faculty) also dead, to the effect that "returned men take off their hats to salute your house"—my own irremovable reward.

And so I conclude with a reference to "the staff." They have been so accustomed to the honour and glory of the situation that I simply present the other side.

Said the Lord Chief Justice of England only a few months ago: "I look askance at the *droit administratif* of bureaucracy and its restless pretensions." He is not alone, and before the end of the war an able publicist penned the following verdict:

> A bureaucracy working without proper external control becomes a prey to red tape and routine officialdom and place hunting and tends to stifle individual initiative and sense of responsibility. In its passion for self-existence it forgets the object of its existence—good government—and in its desire to secure the smooth working of the machine it becomes inhuman and conservative. Above all, it develops a hypersensitiveness to criticism which compels it to do all in its power to crush freedom of speech and freedom of the press.

Is not this too often the case of my critics? Certainly some such display killed Sydney Hubert in the Crimea and maimed Florence Nightingale, even though she had all the weight of Queen Victoria herself behind her. So far as I can see, such spirit and defaults are still a mighty menace, destroying many. And surely one of the greatest problems of the future will be how to increase its powers for good and, at the same time, how to minimize and finally extinguish its almost equal powers for evil.

Yours, etc.,
J. W. SPRINGTHORPE,
Lieutenant-Colonel, A.A.M.C.

32, Collins Street,
Melbourne, C.1.
(Undated.)

[This correspondence is now closed.—EDITOR.]

## Books Received.

THE CASE FOR ACTION, A SURVEY OF EVERYDAY LIFE UNDER MODERN INDUSTRIAL CONDITIONS, WITH SPECIAL REFERENCE TO THE QUESTION OF HEALTH, by I. H. Pearse, M.D., B.S., and G. S. Williamson, M.C., M.D.; 1931. London: Faber and Faber Limited. Crown 8vo., pp. 183. Price: 5s. net.

## Diary for the Month.

APR. 1.—Victorian Branch, B.M.A.: Branch.
APR. 2.—South Australian Branch, B.M.A.: Council.
APR. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
APR. 9.—Victorian Branch, B.M.A.: Council.
APR. 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 10.—Queensland Branch, B.M.A.: Branch.
APR. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
APR. 22.—Victorian Branch, B.M.A.: Council.
APR. 24.—Queensland Branch, B.M.A.: Council.
APR. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 30.—South Australian Branch, B.M.A.: Branch.
APR. 30.—New South Wales Branch, B.M.A.: Branch.

## Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xiv.

ALFRED HOSPITAL, MELBOURNE, VICTORIA: Honorary Vacancies.

MATER MISERICORDIÆ HOSPITAL, NORTH SYDNEY, NEW SOUTH WALES: Honorary Surgeon, Honorary Physician.

NEW SOUTH WALES RUGBY FOOTBALL LEAGUE, SYDNEY: Central Medical Officer.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: General Medical Superintendent, Honorary Consulting Urologist.

THE WOMEN'S HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Clinical Assistant.

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney. | Australian Natives' Association.<br>Ashfield and District United Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham United Friendly Societies' Dispensary.<br>Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney.<br>North Sydney Friendly Societies' Dispensary Limited.<br>People's Prudential Assurance Company, Limited.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members desiring to accept appointment in ANY COUNTRY HOSPITAL are advised to submit a copy of their agreement to the Council before signing, in their own interests.<br>Brisbane Associated Friendly Societies' Medical Institute.<br>Mount Isa Hospital.<br>Mount Isa Mines. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Lodge Appointments in South Australia.<br>All Contract Practice Appointments in South Australia. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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